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Southern Medical Association, Forty-Seventh Annual Meeting, Atlanta, Georgia, October 26-29, 1953

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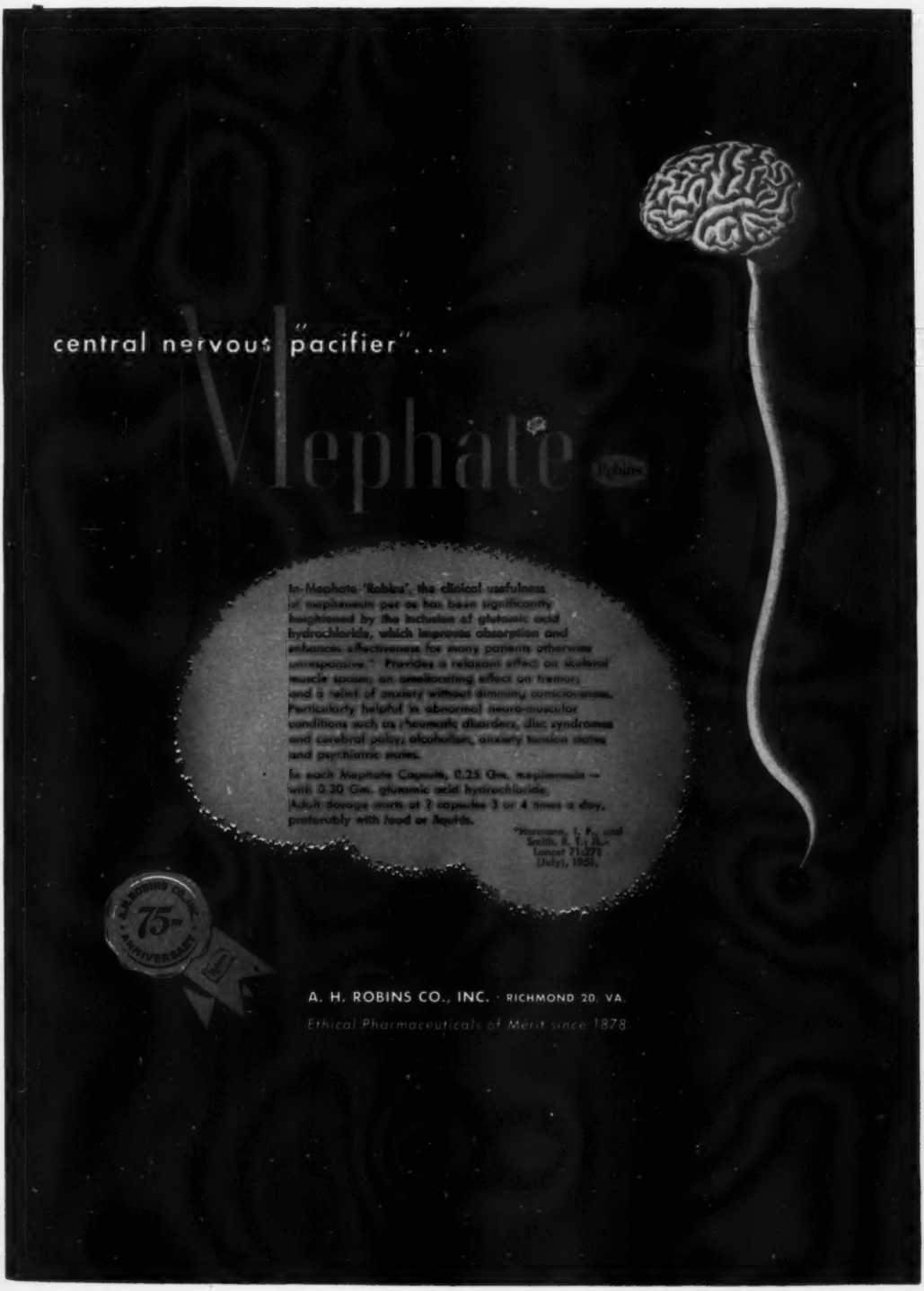
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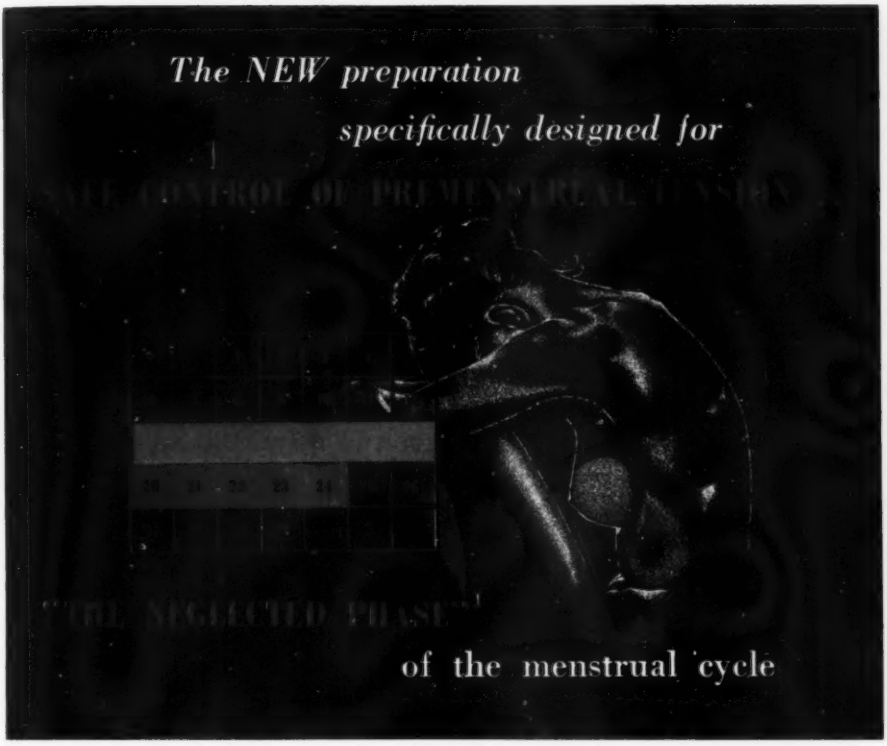

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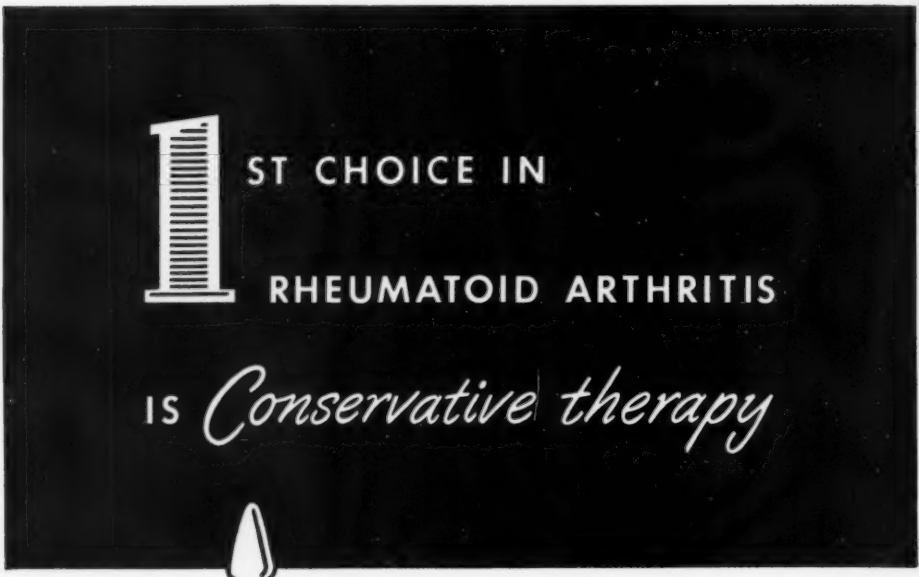

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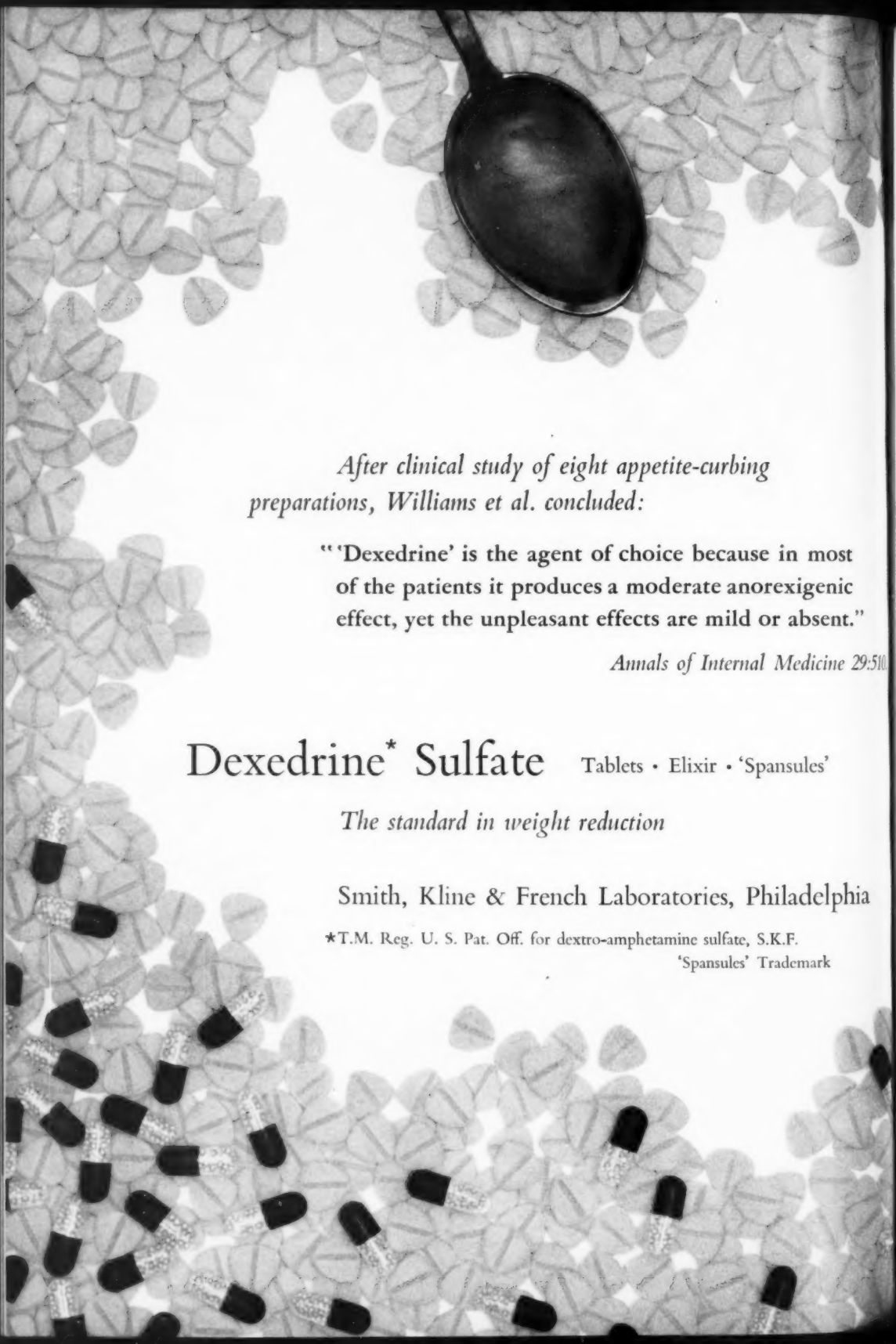
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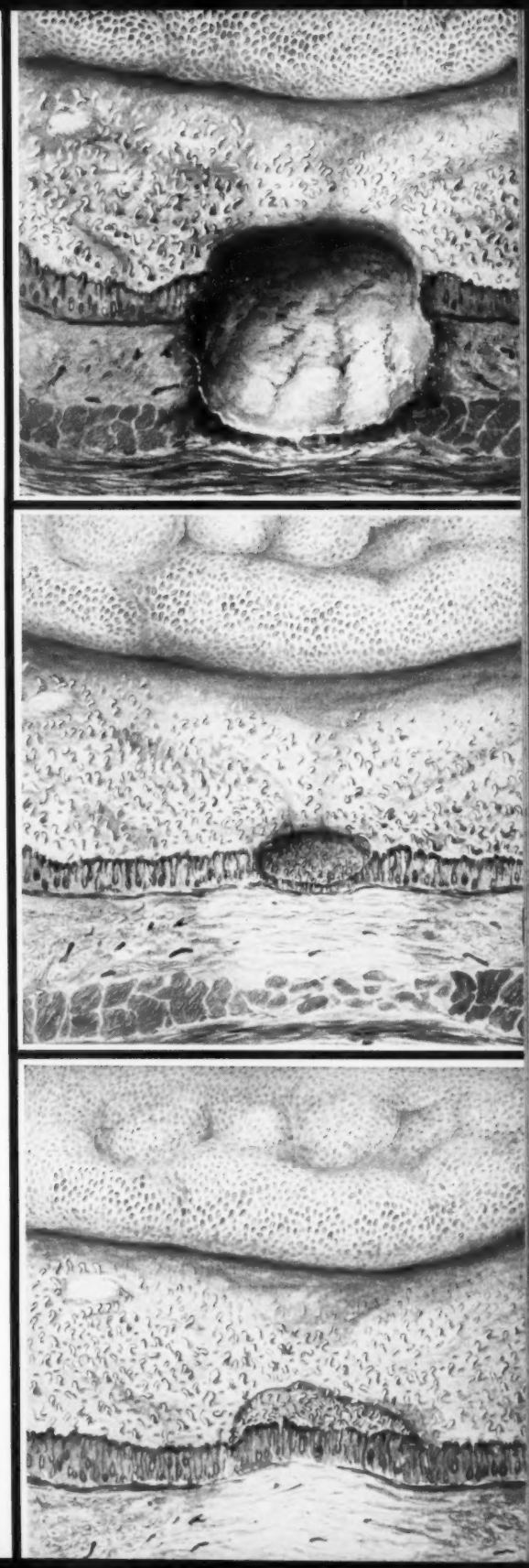

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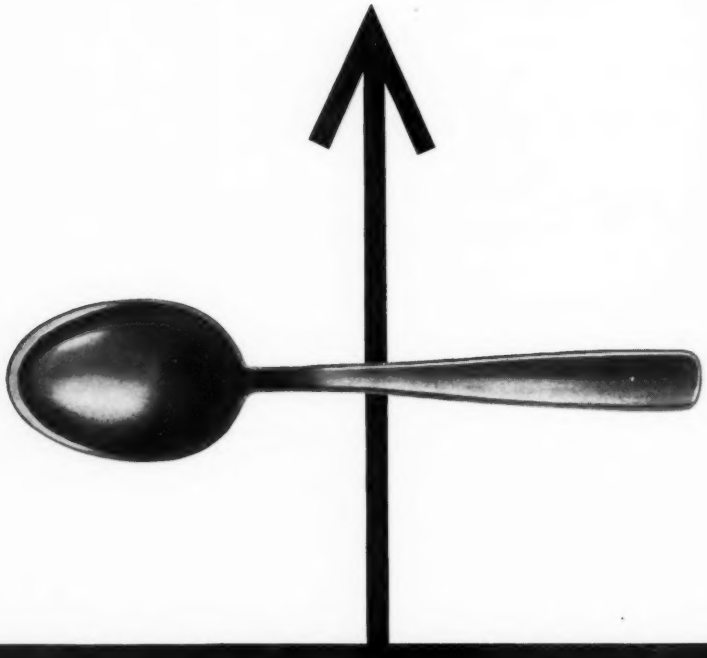

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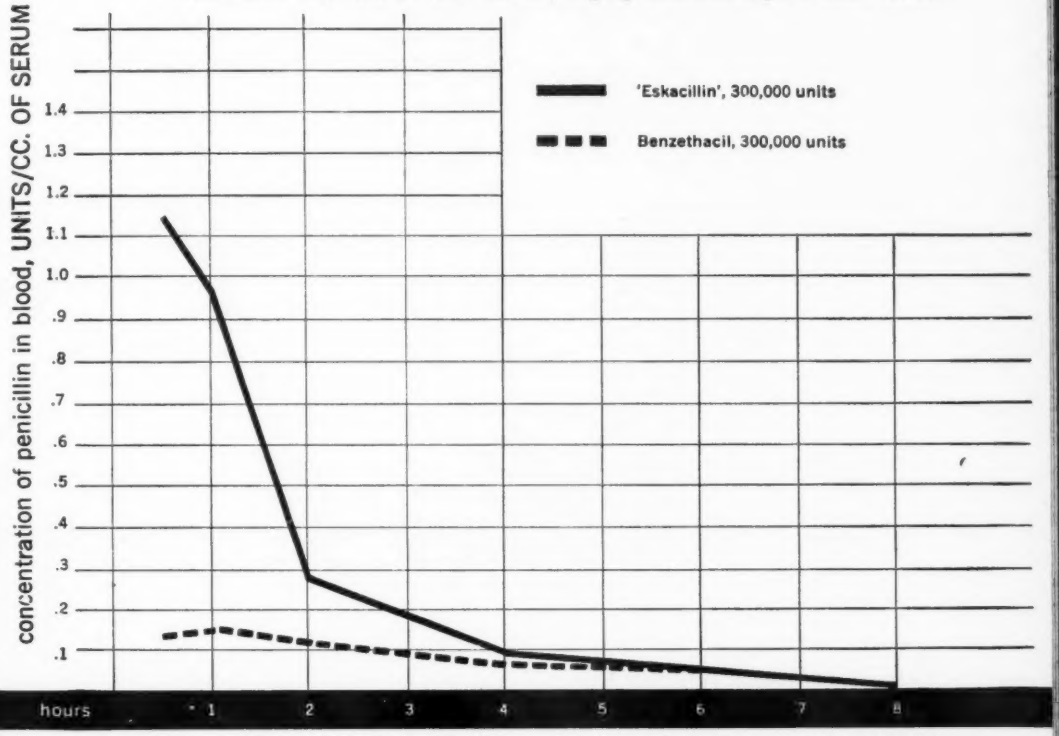

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LOW IN COST TO PATIENTS

Available at all pharmacies

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STUART COMPANY
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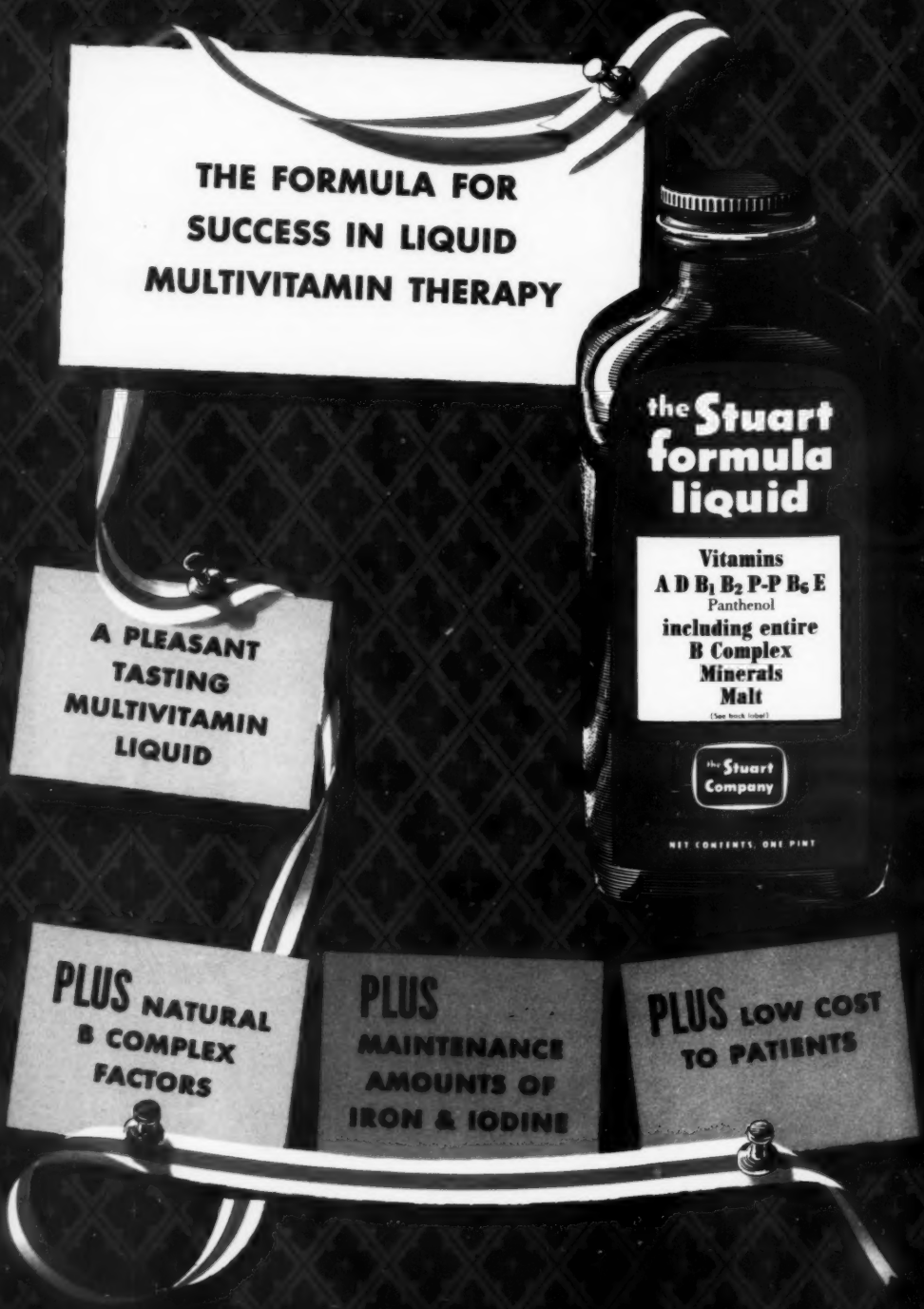
THE FORMULA FOR SUCCESS IN LIQUID MULTIVITAMIN THERAPY
A PLEASANT TASTING MULTIVITAMIN LIQUID
PLUS NATURAL B COMPLEX FACTORS
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including entire
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dermatoses

eczemas

external ulcers

diaper rash

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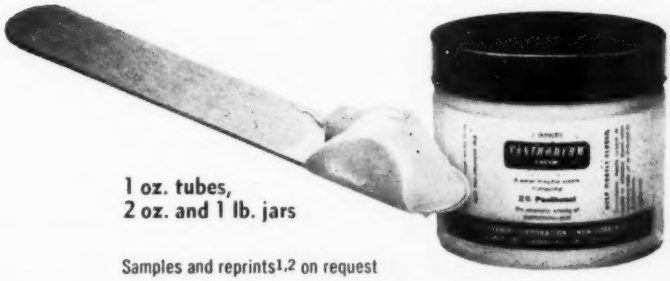
panthoderm
cream

first and **only** topical therapy to contain panthenol

CLINICALLY EFFECTIVE — new studies[1,2] show that topical panthenol (analog of pantothenic acid) "favorably influenced the course of various ulcerative and pyogenic dermatoses. A majority healed and many showed various degrees of improvement." Even long standing conditions resistant to other therapy seem to respond to Panthoderm Cream which . . .

- **relieves pain and itching**
- **promotes granulation and healing**

PLEASANT TO APPLY — non-staining, smooth-spreading; nontoxic, relatively non-sensitizing.



1 oz. tubes,
2 oz. and 1 lb. jars

Samples and reprints[1,2] on request

U. S. VITAMIN CORPORATION
Casimir Funk Laboratories, Inc. (affiliate)
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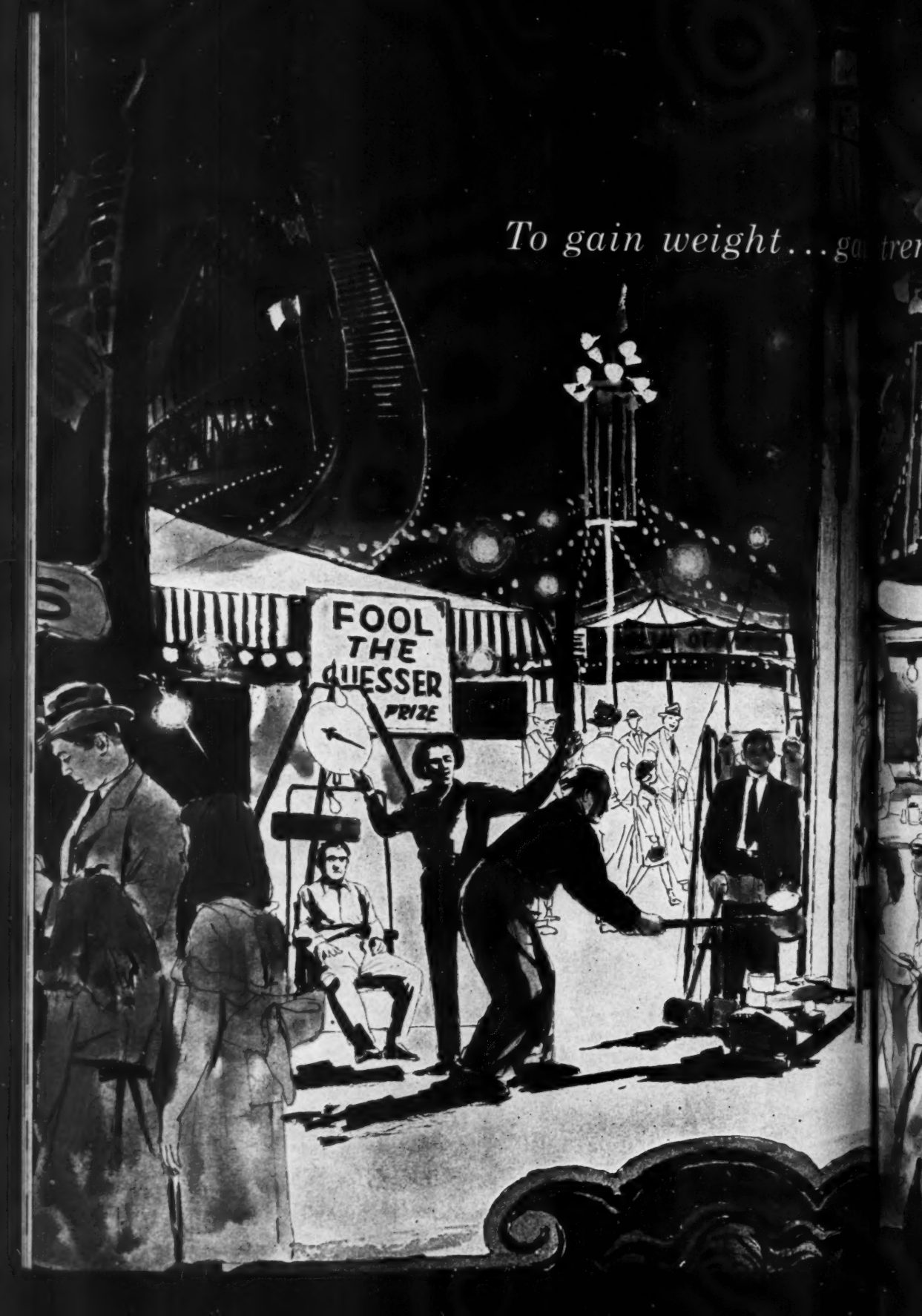
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FOOL
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GUESSER
PRIZE

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[ORAL FAT EMULSION SCHENLEY]

A highly palatable emulsion containing 50 percent coconut oil and 12½ percent sucrose, useful whenever caloric intake must be increased without undue increase in bulk.

Delicious alone, or when taken with milk and other fluids, semisolid foods, and desserts.

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For children, or where fat tolerance is a problem, small initial dosage may be prescribed, then increased to the level of individual capacity.

Available through all pharmacies, in bottles of 16 fl.oz.

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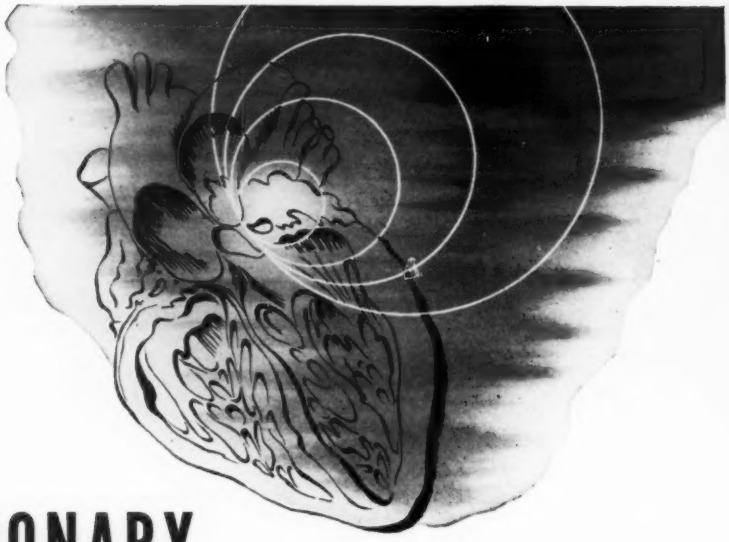

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To improve and strengthen the action of the failing heart through dilating the coronary arteries and to reduce the energy requirements of the heart by mild sedation, are widely desired treatment aims. A great host of physicians recognize theobromine and the sedative, phenobarbital, as admirably suited to these requirements.

Abundant evidence exists that theobromine dilates the coronary arteries. Theobromine also provides safe myocardial stimulation and diuresis. TCS offers the excellent theobromine salicylate, highly efficient because of its extremely high intestinal solubility and absorbability, and uniformly well tolerated because of calcium salicylate, which reduces the gastric solubility of theobromine salicylate.

TCS *tablets*

DOSAGE: One to two tablets 3 to 4 times daily. Reduce with improvement.

SUPPLY: In bottles of 50 and 250 tablets. Each TCS Tablet supplies 6 gr. theobromine salicylate, 1 gr. calcium salicylate and ¼ gr. phenobarbital.

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BASIC
in
Antidiarrheal
Therapy

Arobon®

Whenever diarrhea is encountered, whether in adults, children or infants, and regardless of severity, Arobon is profitably employed as the basic medication. Prepared from specially processed carob flour, Arobon provides generous amounts of naturally occurring pectin, lignin, and hemicellulose. These complex carbohydrates exert the very actions required for prompt control of diarrhea: They are demulcent, adsorbent, soothing, water-binding, antiputrefactive.

In simple diarrhea of adults, infants and children, Arobon usually suffices as the sole medication. In infectious diarrhea and the dysenteries, it is a valuable adjuvant to specific therapy. Arobon is safe, devoid of side actions, and does not interfere with nutrient absorption.

Simple to Prepare Arobon is simply prepared: The powder is merely stirred into milk or water, forming a highly palatable drink. Suggested doses: for children and adults, 1 to 2 level tablespoonfuls in milk or water; for infants, 2 to 4 level teaspoonfuls boiled in water. Although containing no chocolate, the resulting mixture has a palatable, chocolate-like taste acceptable to all patients.

AROBON
is supplied in 5 ounce jars and is available through all pharmacies.

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LIQUID and CAPSULES

for the common hypochromic and hyperchromic anemias; nutritional and megaloblastic anemias of pregnancy, pellagra and sprue.

Provides comprehensive therapy with Iron, Liver and Vitamins, including Vitamin B_{12}.

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specific for intestinal amebiasis

Fumidil

(Fumagillin, Abbott)

a potent, new direct acting antibiotic

FUMIDIL is direct acting

FUMIDIL, unlike the broad spectrum antibiotics, is directly amebicidal. It has no antibacterial spectrum, and does not affect normal flora in the intestinal tract.

FUMIDIL is well tolerated

At the recommended dose, side effects from FUMIDIL have been transient and have rarely been of such significance as to justify the interruption of therapy.

FUMIDIL is specific for amebiasis

FUMIDIL acts specifically against *E. histolytica* without effect upon the bacteria present. Thus, the danger of overgrowth of yeasts and fungi is slight. Reported studies show low incidence of recurrence.

FUMIDIL is effective orally

Recommended dosage for the average adult is 30 to 60 mg. daily, in divided doses, three or four times a day for 10 to 14 days. One course usually clears most cases. In 10-mg. capsules, bottles of 30.

For more information, write Abbott Laboratories, North Chicago, Illinois. Abbott

1-132

free flow

IN URINARY TRACT INFECTIONS

Free flow of urine is essential to successful chemotherapy. Thus, it is important to select the sulfonamide preparation which is least likely to produce crystalluria.

On the score of both potency and solubility in acid urine, SULFOSE is unsurpassed.

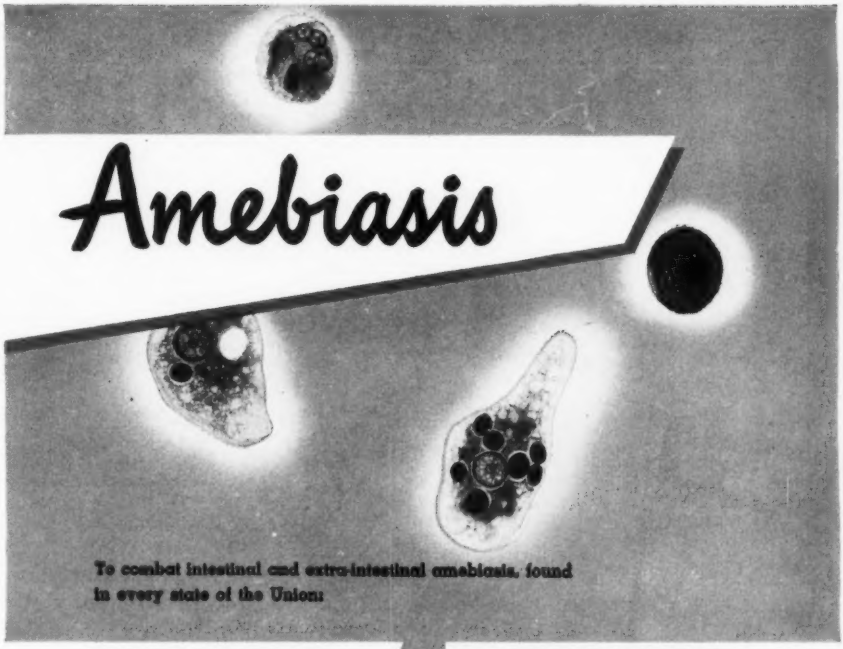
SULFOSE®

Triple Sulfonamides Wyeth

ORAL SUSPENSION **TABLETS**

Wyeth®

Philadelphia 2, Pa.



Amebiasis

To combat intestinal and extra-intestinal amebiasis, found in every state of the Union:

MILIBIS®

because of relative insolubility, assures high concentration in the large intestine, very effective against subacute and chronic amebiasis. Average adult dose: 0.5 Gm. (1 tablet) three times daily for 7 to 10 days, repeated if necessary. Control acute dysentery first or concurrently with emetine.

Supplied in 0.5 Gm. tablets, bottles of 25.

ARALEN® DIPHOSPHATE

—the well known antimalarial— induces *complete clinical remission* in pleuropulmonary amebiasis[1] as well as hepatic and other forms of extra-intestinal amebiasis.[2,3] Average adult dose: 1 Gm. (4 tablets) daily for 2 days, then 0.5 Gm. daily for 2 to 3 weeks, which may be combined with or successive to Milibis therapy of intestinal amebiasis.

Supplied in 0.25 Gm. tablets, bottles of 100 and 1000.

Winthrop-Stearns INC.
NEW YORK 18, N. Y. WINDSOR, ONT.

Milibis and Aralen, trademarks reg. U. S. & Canada, brand of bismuth glycolylarsanilate and chloroquine, respectively.

1. Lindsay, A. E., Gossard, W. H., and Chapman, J. S.: Dis. Chest, 20:383, Nov., 1951.
2. Caven, N. J., Jr.: Am. Jour. Med., 6:327, Mar., 1949.
3. Emmott, J.: J.A.M.A., 141:22, Sept. 3, 1949.

Illustrated brochure on request.

the ***most widely used***

ethical specialty for

care** of the **infant's skin

DESITIN®

OINTMENT

the pioneer external cod liver oil therapy

Decisive studies[1,2] substantiate over 25 years of daily clinical use regarding the ability of Desitin Ointment to . . . . . *protect, soothe, dry and accelerate healing in . . .*

- **diaper rash** • **exanthema**
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- **intertrigo** • **prickly heat**
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(due to urine, excrement, chemicals or friction)

Desitin Ointment is a non-irritant blend of high grade, crude Norwegian cod liver oil (with its unsaturated fatty acids and high potency vitamins A and D in proper ratio for maximum efficacy), zinc oxide, talcum, petrolatum, and lanolin. Does not liquefy at body temperature and is not decomposed or washed away by secretions, exudate, urine or excrements. Dressings easily applied and painlessly removed.

Tubes of 1 oz., 2 oz., 4 oz., and 1 lb. jars

write for **samples** *and literature*

DESITIN CHEMICAL COMPANY

70 Ship Street • Providence 2, R. I.

1. Heimer, C. B., Grayzel, H. G. and Kramer, B.: Archives of Pediat. 68:382, 1951.
2. Behrman, H. T., Combes, F. C., Bobroff, A. and Leviticus, R.: Ind. Med. & Surg. 18:512, 1949.

New, Improved 'Homicebrin' with Vitamin B_{12}

'Homicebrin' now provides *six* essential vitamins in a smooth, palatable, homogenized mixture.

Each 5 cc. contain:

Vitamin A (Palmitate)	3,000 U.S.P. units
Thiamin Chloride	1 mg.
Riboflavin	1.2 mg.
Vitamin B_{12} (Activity Equivalent)	3 mcg.
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Prescribe the 4-ounce or the economical 1-pint bottle. There is no increase in price.

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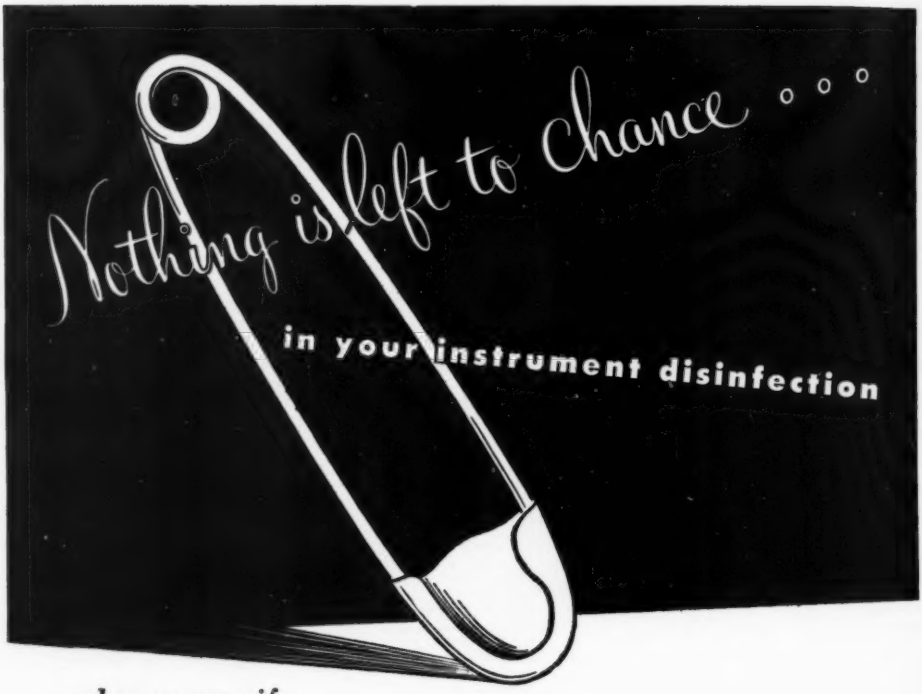
Lilly

For the prophylaxis or treatment of multiple vitamin deficiencies in infants and children.

'Homicebrin'

(HOMOGENIZED MULTIPLE VITAMINS, LILLY)



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BARD-PARKER FORMALDEHYDE GERMICIDE

containing HEXACHLOROPHENE (G-11*)

• • • because it has established a new standard of potency for solutions used in the chemical disinfection of surgical instruments. It will destroy vegetative pathogens and spore formers within 5 minutes, and the spores themselves within 3 hours—as shown in the comparative chart. In addition, it is "economically usable" as prolonged immersion of delicate steel instruments will not result in rust or corrosive damage to keen cutting edges. The Solution will retain its high disinfecting potency over long periods of use if kept undiluted and free of foreign matter. *Trademark of Sindar Corp.

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For practical purposes we suggest the selection of B-P CONTAINERS — all scientifically designed for use with the Solution.

Compare this significant data evaluating the potency of the IMPROVED germicide

SPORULATING BACTERIA	50% DRIED BLOOD	WITHOUT BLOOD
Cl. tetani	3 hours	3 hours
Cl. welchii	2 hours	2 hours
B. anthracis	1½ hours	1½ hours
VEGETATIVE BACTERIA		
Staph. aureus	5 min.	15 sec.
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Strept. hemolyticus	2 min.	15 sec.

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RHINALGAN®

is a *Safe* Nasal Decongestant

IN PEDIATRICS
IN GERIATRICS

It does not affect
BLOOD PRESSURE
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NOT required to have the stigma
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It is *Safe* in
CARDIAC · DIABETIC
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ENTIRELY FREE OF SIDE-EFFECTS . . . no cumulative action . . . no overdosage problem . . . acts quickly . . . effective for prolonged periods . . . non-toxic . . . pleasant . . . efficient.

Supplied in: THE DOHONY SPRAY-O-METER (Combination Spray and Dropper)

Also for Office and Hospital use in: Vial bottles (Dropper or Spray)

Available on YOUR prescription only!

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INFANTS—as spray or drops.

Formula: Desoxyephedrine Saccharate 0.50% w/v in an isotonic aqueous solution with 0.02% Laurylcamphonium saccharin. Flavored. pH 6.4.

REFERENCES:
1. Van Alyea, O. E., and Donnelly, W. A.: E.E.N. & T. Monthly, 31: Nov. 9, 1952.
2. Fein, E. L.: AMA Arch. Otolaryn., 57: 607-609, 1953.
3. Mortimer, N., and Hadley, A.: N.Y. Phys., 26, 14-15, 1950.
4. Dow, J. E. (as CAI, HC-USAF): Research Report, Dept. Otolaryn., USAF School Aviat. Med., 1952.
5. Hamilton, W. F., and Turnbull, F. W., Jr.: Amer. Physiol. Arch., No. 7, 378-382, 1950.

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after 40 years—STILL the auralgesic and decongestant

RECTALGAN-Liquid for Symptomatic Relief in Hemorrhoids, Proctitis, Perineal Suturing

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Double scored 0.5 Gm. tablets.
Bottles of 100 and 1000.

Syrup (0.25 Gm. Elkosin per 4 cc.), microcrystalline suspension in strawberry-flavored vehicle.
Bottles of 16 fluidounces.

- Remarkably low incidence of side effects — less than 5%
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- Renal complications rare
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Ciba PHARMACEUTICAL PRODUCTS, INC., SUMMIT, N. J. 2-1690M

for RAPID
SUSTAINED
CONTROLLABLE
REDUCTION OF
Critically Elevated
Blood Pressure

THE PARENTERAL SOLUTIONS OF

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SOLUTION
INTRAMUSCULAR VERILOID

Injected deep into the muscle, a single dose attains its maximum hypotensive response in 60 to 90 minutes. By repeated injection every 3 to 6 hours, the blood pressure may be kept depressed for hours or days if necessary. Solution Intramuscular Veriloid provides 1.0 mg. of alkavervir (mixed Veratrum viride alkaloids) per cc. of isotonic buffered aqueous solution.

SOLUTION
INTRAVENOUS VERILOID

Given in proper dilution slowly by vein, Solution Intravenous Veriloid usually reduces both the systolic and diastolic blood pressures in a matter of minutes—entirely within the control of the physician. This valuable emergency drug frequently proves to be a life-saving measure. Contains 0.4 mg. of alkavervir (mixed Veratrum viride alkaloids) in 0.25 per cent acetic acid solution.

Acting rapidly in a significantly high percentage of patients, the parenteral solutions of Veriloid afford a positive means of reducing critically elevated blood pressure. They have been found of great value for the relief of the distressing symptoms of prolonged, severe blood pressure elevation, and in hypertensive emergencies when the degree of hypertension assumes life-threatening proportions. These parenteral agents are indicated in hypertensive states accompanying cerebral vascular disease, malignant hypertension, hypertensive crisis (encephalopathy), toxemias of pregnancy, pre-eclampsia, and eclampsia.

RIKER LABORATORIES, INC.
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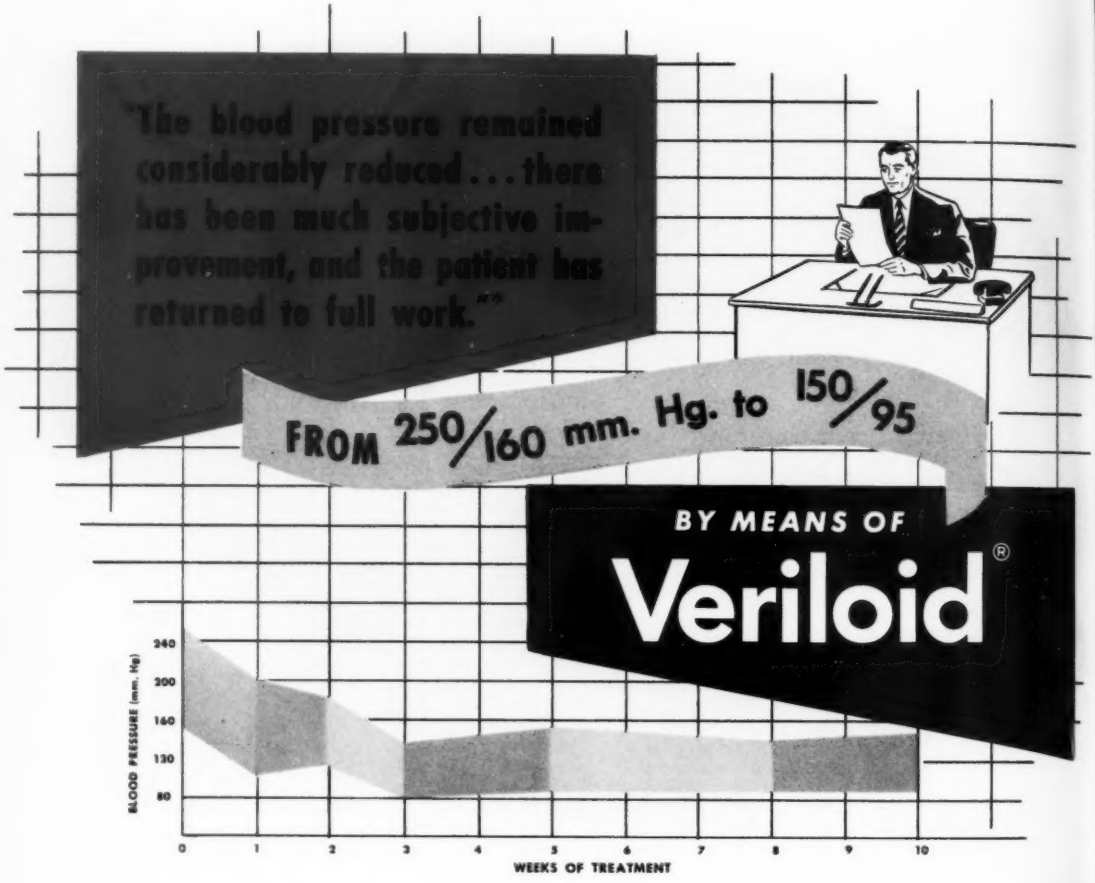
VERILOID, GENERICALLY DESIGNATED ALKAVERVIR, IS

An Original Riker Research Product



Veriloid is available in three dosage forms:

Veriloid (plain) in 1, 2, and 3 mg. scored tablets; starting dosage 9 to 15 mg. daily, to be adjusted according to response and tolerance.

Veriloid with Phenobarbital (Veriloid-VP), each scored tablet presenting Veriloid 2 mg. and phenobarbital 15 mg.

Veriloid-VPM, each scored tablet containing Veriloid 2 mg., phenobarbital 15 mg., and mannitol hexanitrate 10 mg. Initial recommended dosage for VP and VPM, 1 to 1½ tablets t.i.d. or q.i.d.

This male patient, 41 years of age, was given the same dose of Veriloid tablets (26 mg. daily) for a period of five months. During that time, he was able to resume his normal sedentary occupation at full activity. Note the prompt and sustained reduction in diastolic pressure and the subsequent good reduction in systolic pressure.

Not every patient shows this spectacular response to oral Veriloid. However, a sufficiently large number do, warranting the administration of this hypotensive agent to every patient with elevation of blood pressure sufficient to require treatment.

*Kauntze, R., and Trounce, J.: Treatment of Arterial Hypertension with Veriloid, Lancet 2:1002 (Dec. 1) 1951.

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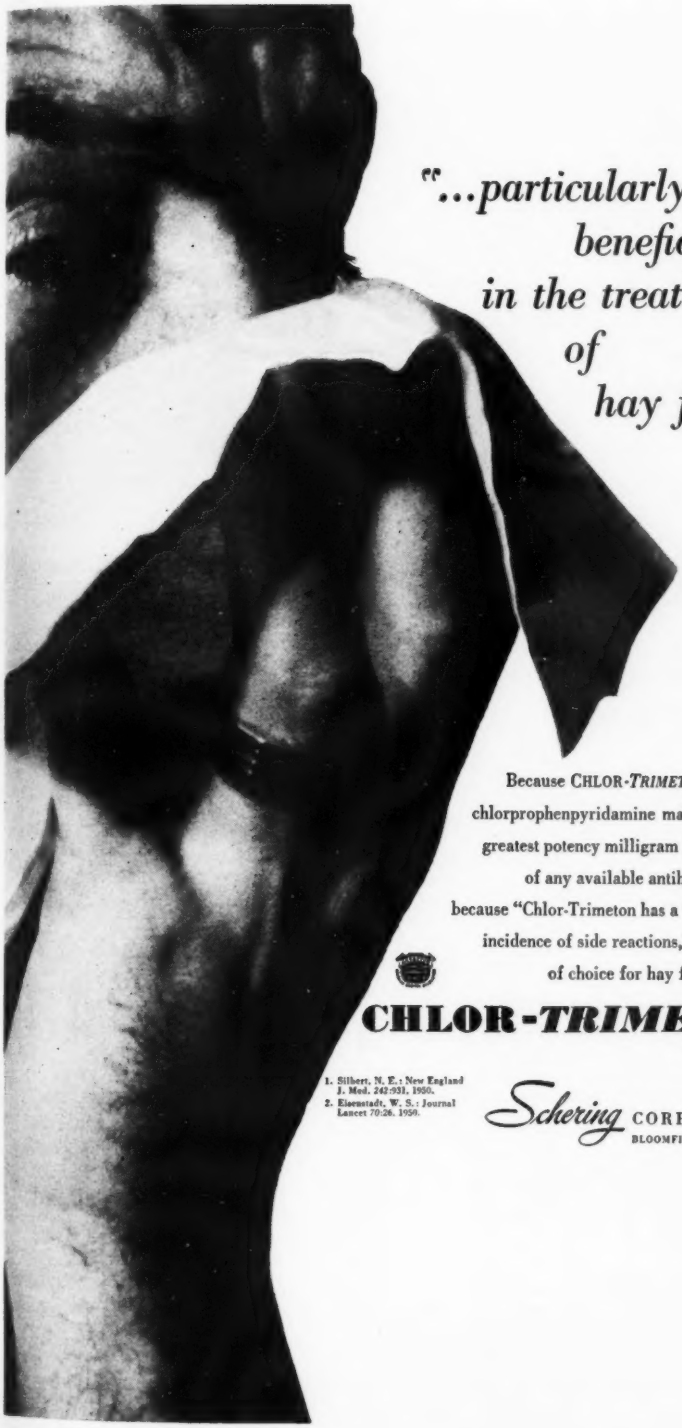

"...particularly beneficial in the treatment of hay fever."[1]

Because CHLOR-*TRIMETON*® maleate, chlorprophenpyridamine maleate, has the greatest potency milligram for milligram of any available antihistamine, and because "Chlor-Trimeton has a relatively low incidence of side reactions,"[2] it is a drug of choice for hay fever patients.

CHLOR-*TRIMETON*
maleate

1. Silbert, N. E.: New England J. Med. 242:931, 1950.
2. Eisenstadt, W. S.: Journal Lancet 70:26, 1950.

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CHLOR-TRIMETON

Top Flushing Efficiency

plus smooth muscle relaxation in biliary tract disorders with

Cholan *hmb*®

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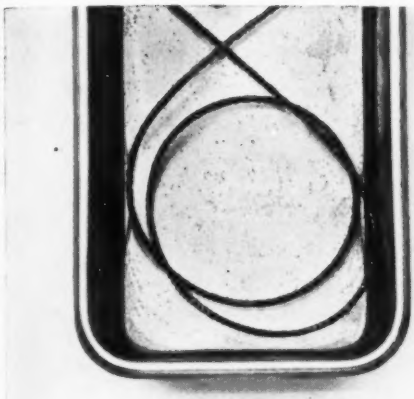
Maltbie . . . first to develop American process for converting crude viscous ox-bile into chemically pure dehydrocholic acid

Each tablet contains: **Dehydrocholic acid Maltbie.....250 mg. (3¾ gr.)**
Homatropine methylbromide.....2.5 mg. (1/24 gr.)
Phenobarbital.....8 mg. (⅛ gr.)

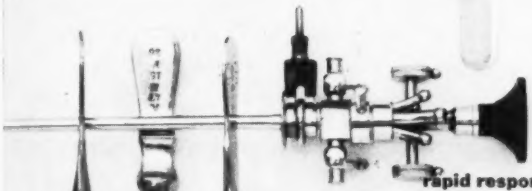
COMPARISON SHOWING ++++ EFFICIENCY OF Cholan *hmb*

TYPICAL AGENT	CLASSIFICATION	EFFECT	FLUSHING* EFFICIENCY	COMMENT
Fats or natural bile salts	Cholagogue	Promotes evacuation of the gallbladder	+	Utilizes only bile of normal viscosity present in the gallbladder
Natural bile or unconjugated bile salts	Choleretic	Stimulates secretion of normal bile by the liver	++	Utilizes increased amounts of bile of normal viscosity
Dehydrocholic acid	Hydrocholeretic	Stimulates secretion of fluid bile by the liver	+++	Utilizes copious amounts of free-flowing bile – adequate in absence of spasm of sphincter of Oddi
Dehydrocholic acid, homatropine methylbromide, phenobarbital (CHOLAN HMB)	Hydrocholeretic, parasympatholytic, sedative	Stimulates secretion of fluid bile by the liver, and relaxes sphincter of Oddi spasm	++++	Utilizes copious amounts of free-flowing bile and relaxes smooth muscle spasm for greater therapeutic efficacy



read Pfizer Spectrum weekly in the J.A.M.A.

IN URINARY TRACT INFECTIONS



rapid response

"Patients with pyelitis were well and doing their usual duties within 24 hours . . ."[1] ". . . resistant cases showed remarkable response."[2]

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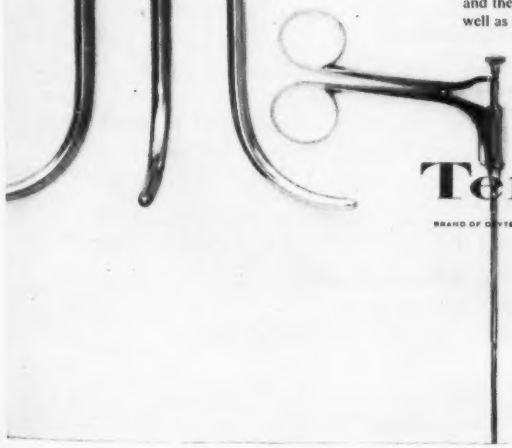
"Terramycin was selected . . . in view of high urinary excretion rate following small oral doses of the antibiotic."[1]

unexcelled toleration

"Terramycin is generally well tolerated, the percentage of relapses being low and the percentage of bacteriological as well as clinical cures high."[3]

1. Canad. M. A. J. 66:151 (Feb.) 1952.
2. J. Urol. 67:762 (May) 1952.
3. Ibid. 69:315 (Feb.) 1953.



Terramycin

BRAND OF OXYTETRACYCLINE

Pfizer

Introducing a new Valentine product...

for more effective control of gastric hyperacidity.

Particularly indicated in peptic ulcer, "heartburn" of pregnancy, gastric hypermotility, chronic dyspepsia and other functional indigestions.

acichek

VALENTINE

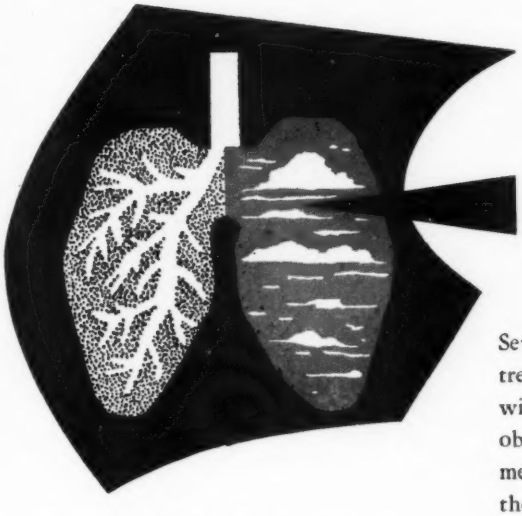
A combination of dihydroxy aluminum aminoacetate, N.N.R., sodium carboxymethylcellulose and glycine.

Supplied in bottles of 100 and 1000 tablets.

- acts almost immediately to give prompt relief from gastric distress
- maintains a desirable pH of gastric contents for hours with no depression of peptic activity
- produces neither secondary acid rise nor systemic alkalosis
- provides a mild, physiologic corrective of constipation
- easily and acceptably administered in tablets which require no chewing

VALENTINE COMPANY, INC.
Richmond 9, Virginia

The most significant result in the treatment of *Bronchial Asthma*



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(IN GELATIN)

Advantages

Administered as Easily as Insulin:
Subcutaneously or intramuscularly with a minimum of discomfort.

Fewer Injections:
One or two doses per week in many instances.

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Combines the two-fold advantage of sustained action over prolonged periods of time with the quick response of lyophilized ACTHAR.

Much Lower Cost:
Recent significant reduction in price, and reduced frequency of injections, have advanced economy of ACTH treatment.

Severe bronchial asthma can now be treated in the home and in the office with a degree of success similar to that obtained with hospital care. Improvement is prompt and dramatic. Neither the patient's age nor the chronicity of the asthmatic condition detracts from the efficacy of ACTHAR treatment, which has stood the most severe of all tests of usefulness—the requirements of the general practitioner. The use of the disposable cartridge syringe—an immediately available form of HP* ACTHAR *Gel*—can be a life-saving measure in the medical emergency which suddenly arises in the course of long-standing "intractable" asthma. HP*ACTHAR *Gel* has demonstrated its superiority over customary measures in many instances of bronchial asthma, and has brought about gratifying remissions lasting as long as 18 months.

*Highly Purified. ACTHAR® is The Armour Laboratories Brand of Adrenocorticotropic Hormone—ACTH (Corticotropin).

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world-wide dependability

PHYSIOLOGIC THERAPEUTICS THROUGH BIORESEARCH

for prompt
and effective
relaxation
of spasm

Barbidonna®

antispasmodic
and sedative

Barbidonna provides "the wide range of therapeutic usefulness which characterizes the naturally occurring belladonna alkaloids",* plus the calming influence of phenobarbital.

Barbidonna's action is uniform and dependable since the alkaloid content is always constant, in fixed and scientifically apportioned amounts.

Barbidonna is supplied as either
TABLETS or ELIXIR

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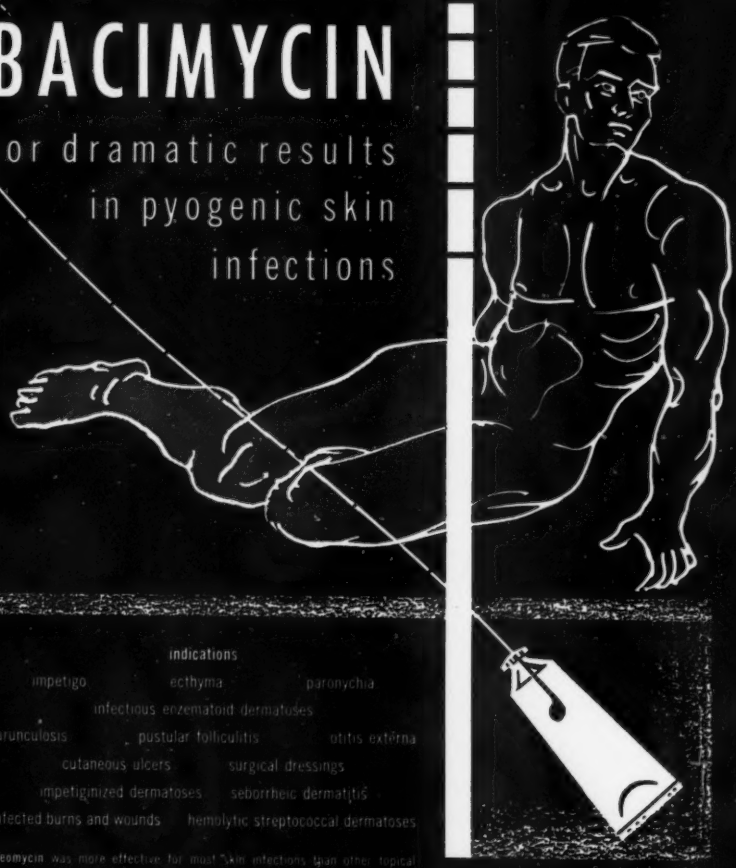

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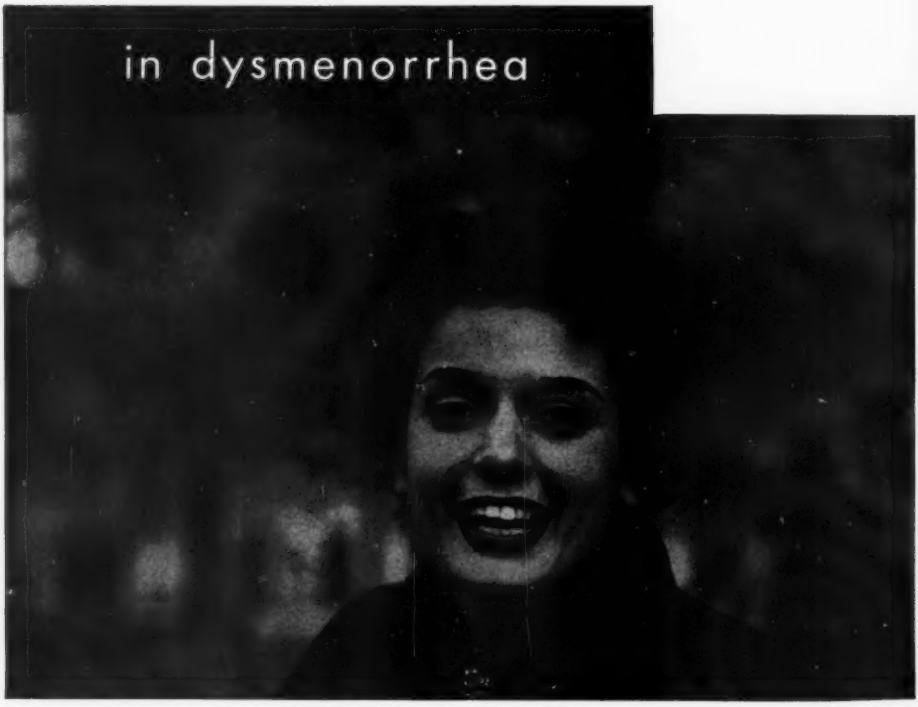

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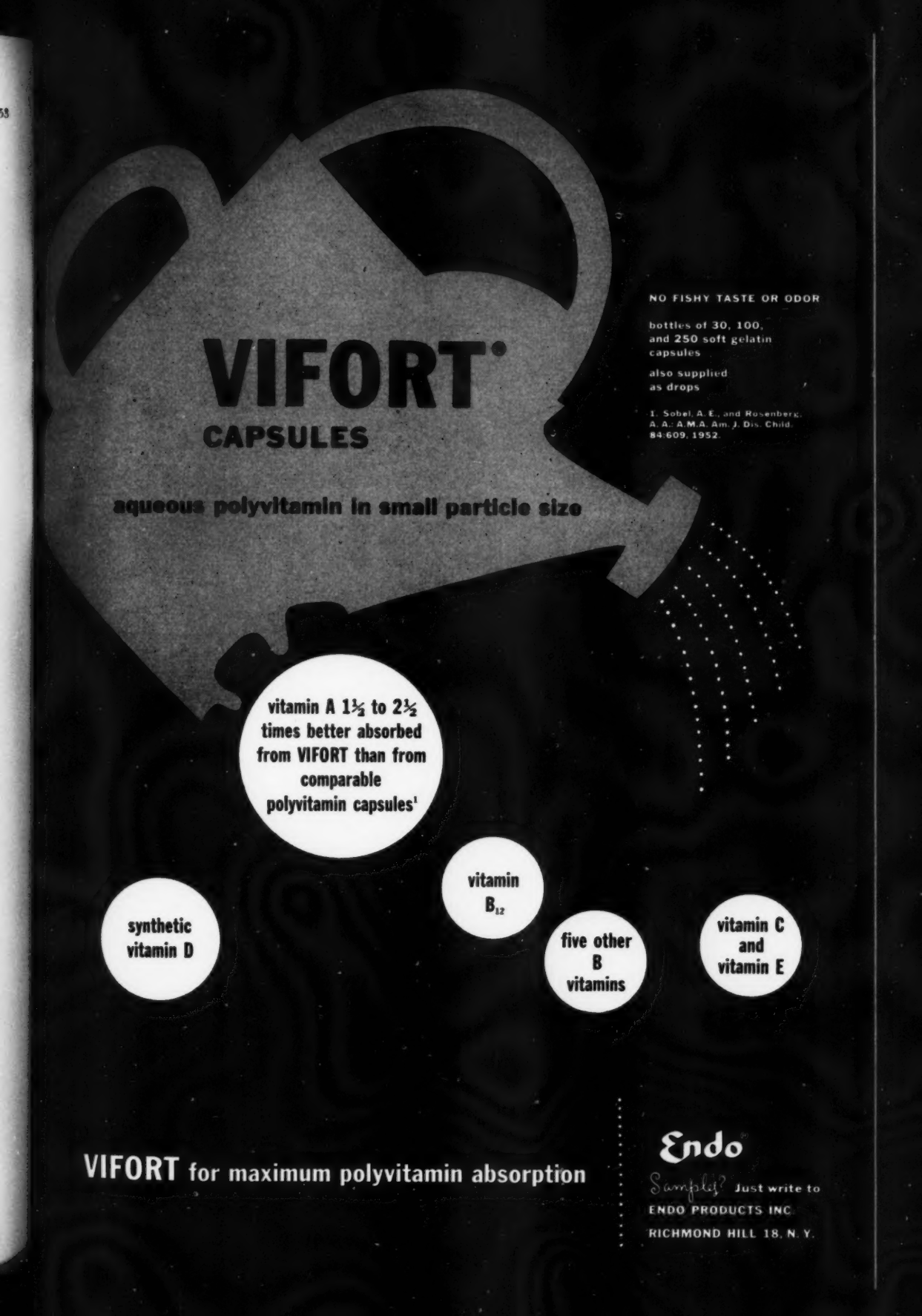
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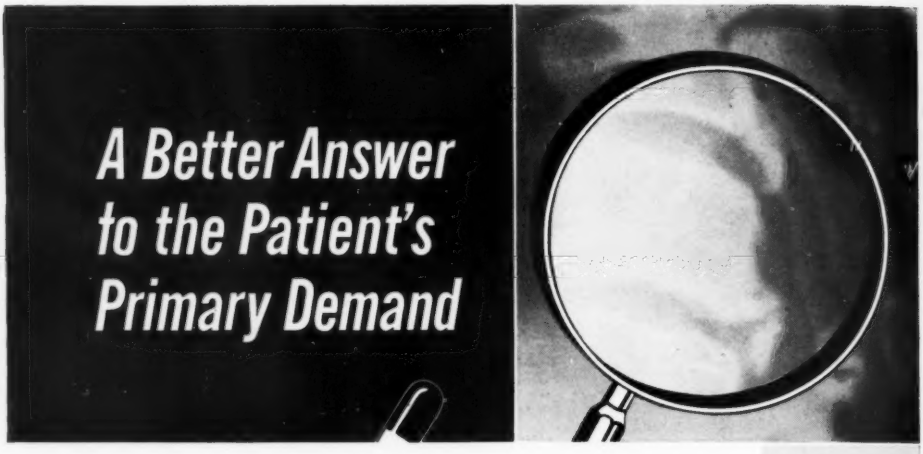

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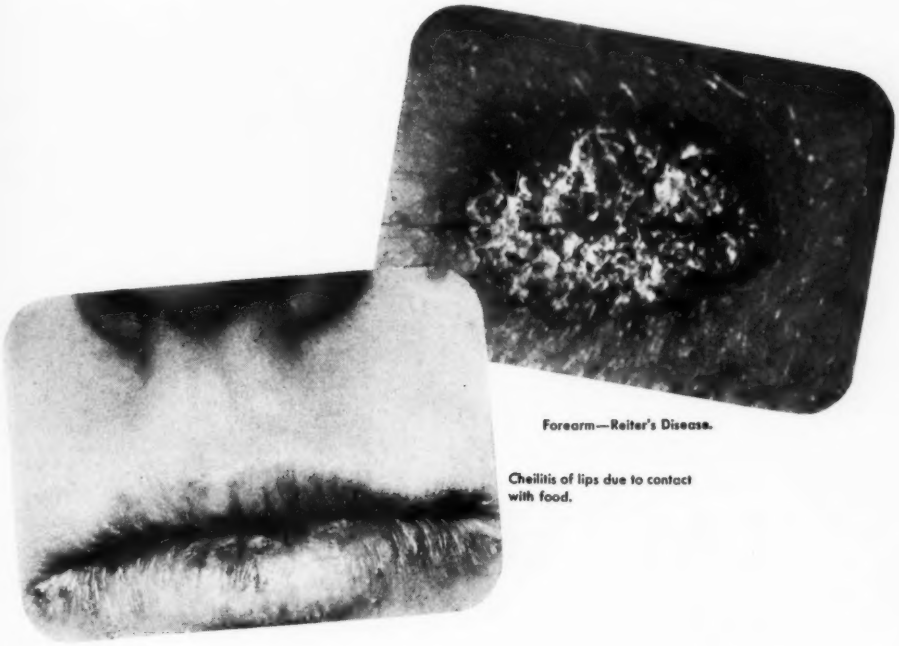

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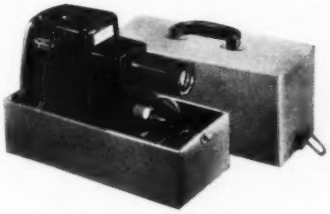

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Volume 46 APRIL 1953 Number 4

DOUBLE CONTRAST STUDIES OF THE COLON: POLYPS IN CHILDREN*

By CHARLES W. YATES, M.D.
Houston, Texas

Although the double contrast technic is the cardinal method for detection of intraluminal lesions of the colon, there seems to be an apparent reluctance on the part of roentgenologists in general to make use of the technic in examining the colon in small children. From discussions with men from various parts of the country, I gather that such reluctance is part of a general distrust of the technic, based on unhappy occurrences from superficial experiences with the technic with adults. I have been rather amazed at some of the expressions concerning the difficulties and hardships encountered in performing the examination. Actually, with a little experience, the examination is only slightly more difficult and time consuming than the barium enema. Such difficulties are easily outweighed by the benefit offered by the technic.

Etymologically, the term "polyp" refers to any pedunculated growth from a mucous surface and in the strictest sense is a gross anatomic designation without regard to the histologic or pathologic character of the tumor.

Through common usage the terms, polyp and polyposis of the colon have come to be used interchangeably with adenoma and adenomatosis. However, polyps of the colon may arise from any of the various elements forming the wall of the colon and will vary histologically accordingly.

Multiple etiologic theories have been offered, most of which fail to satisfy all factors. However, in view of the experiences recorded in the past few years, polyps occurring in the terminal bowel, the majority of which are adenomatous in character, are undoubtedly true tumors. The heredofamilial type of polyposis has been described as a distinct disease entity and the tumors differ from the isolated polyps not in the histological structure but in the number and distribution of the lesions, the history of occurrence in succeeding members of a family and the tendency which has been described as being handed down from one generation to another according to mendelian law. The frequent occurrence of malignancy at an earlier age appears to occur in the heredofamilial type. The "pseudopolyps" occurring with chronic ulcerative colitis have been shown to be isolated islands of mucous membrane of granulations.

In children the adenomatous lesions, on microscopic examination, present a gland pattern which always seems to be well preserved and appears like normal colon mucosa. Goblet cells are very numerous and cell nuclei are small. In adults about one-half have the same appearance as those in children. The other half have fewer goblet cells, nuclei tend to be larger and gland reproduction is not accomplished so well. In a certain few, anaplastic changes in the epithelium are present but no invasion is present.

Isolated adenomatous polyps in children, in spite of their similarity in microscopic appearance, show two distinct variations in characteristics from similar polyps in adults: (1) the relative infrequency of malignant change, and (2) the apparent frequency of self-amputation.

Although the tendency for malignant change is not so great in polyps in children, the possibility cannot be dismissed. There is no single criterion which can be accepted as a reliable index for the malignant potentiality of any given polyp. In polyps of the heredofamilial type, malignant change has been demonstrated as occurring frequently and at fairly early ages.

Self-amputation occurs rarely in polyps in adults. In one hundred cases of polyps in

*Read in Section on Radiology, Southern Medical Association, Forty-Sixth Annual Meeting, Miami, Florida, November 10-13, 1952.

FIG. 1

Routine projection showing anteroposterior appearance of lesion.

FIG. 2

Routine projection showing posteroanterior appearance of lesion.

FIG. 3

Routine projection showing supine oblique appearance of lesion.

FIG. 4

Routine projection showing supine oblique appearance of lesion.

FIG. 5

Bardex® removed to show rectal polyp.

FIG. 6

Prone oblique showing polyp not visualized well on routine projection.

children, Kerr demonstrated self-amputation in eighteen. One wonders if this phenomenon is connected in any way with the relative infrequency of demonstrable polyps between the ages of twelve and twenty years. In reviewing my cases, I am unable to find a case within that age group.

The symptoms associated with polyps of the colon in children appear to be rather constant: (1) blood in the stools occurs in practically one hundred per cent of the cases, and (2) protrusion, diarrhea and abdominal cramps appear in varying degree depending somewhat on the location of the polyps.

Appearance of new polyps probably occurs in approximately 10 per cent of the cases after removal of existing polyps.

Diagnosis.—With symptoms of blood or blood and mucus in the stools, a child should be subjected to both proctoscopic and x-ray examination, both of which should be repeated on recurrence or continuation of symptoms following therapy.

X-ray examination cannot compete with direct visualization and the roentgenologist is doing himself, the patient and the referring physician an injustice if he attempts to accept responsibility for diagnosis of lesions within reach of the proctoscope. However, if suggestion of a lesion is encountered in this region during a colon examination, attention should be called to it.

The larger polypoid lesions in children are probably more easily visualized at fluoroscopy than similar lesions in an adult, but lesions one centimeter or less in diameter will usually be missed with the barium enema. The lesions of smaller size can frequently be visualized with the double contrast technic.

Good cleansing of the colon in children prior to examination is as great a problem as in adults, if not greater. Castor oil is the cathartic of choice and soap suds enemas have offered, in my experience, more than the saline, plain water or other types of enemas.

Re-examination should be carried out in all cases where the bowel is not adequately cleansed, where there is any doubt as to the nature of suspicious defects and where operation is delayed any appreciable length of time following the discovery of a polyp.

Our routine pre-examination preparation is as follows:

(1) No solid supper on the evening preceding examination. Non-curd forming liquids may be taken orally until 9 p.m.

(2) At 7:30 p.m. two ounces of castor oil to children two years or older; one ounce of castor oil to younger individuals.

(3) At 6 a.m. the morning of the examination a soap suds enema is given at fifteen-minute intervals until three enemas have been given. Soap is used as the lubricant for the enema tip.

(4) The patient may have a small breakfast within one hour of the appointment for examination, although, if the right colon is the center of interest, it is probably best not to allow breakfast.

At the time of examination an explanation of the procedure is offered the patient if he or she is old enough for any understanding. Thus far, I have not had to resort to pre-examination sedation in any case.

A size 18 F. Bardex colon tube is used routinely. The bag is inflated under fluoroscopic control. The barium mixture, a preparation of barium sulphate containing a ready-mixed suspending agent which has been weighed and added to a measured volume of water, is allowed to flow to the midportion of the transverse colon and is carried from there to the region of the hepatic flexure by tilting the table and rotating the patient. During the entrance of the barium suspension, the usual fluoroscopic observations and manipulations are carried out, stopping the flow of barium at any point if more than ordinary time is to be used in palpation. The excess barium suspension is siphoned off by lowering the enema can, tilting the table and massaging the abdomen along the course of the colon as necessary. Air is then injected under fluoroscopic control, tilting the table and rotating the patient as necessary to distribute the barium to the right colon. The colon must be well distended for the best visualization.

Following the injection of air, radiographs must be made as quickly as possible to prevent drying of the medium on the colon wall. The use of high kilovoltage and milliamperage to allow the shortest possible exposure time is essential for good quality films.

Routinely, we use four projections: anteroposterior, posteroanterior and both supine obliques *(Figs. 1, 2, 3* and *4)*. The different projections allow the barium remaining in the colon to shift, usually clearing all parts of the colon for visualization. Occasionally, additional views are made if suggested by fluoroscopic observations *(Figs. 5* and *6)*. It must be remembered that speed is essential and the technic does not allow for satisfactory repetition without repeating pre-examination preparations.

FIG. 7

Two polyps in colon measuring 5 millimeters in diameter diagnosed by double contrast study prior to, and confirmed by, surgery.

Although less cooperative than most adults, children as a whole accept the procedure very well. The greatest difficulty I have experienced during colon examinations in children has been during the palpation of the colon while fluoroscoping, and this can be reduced to a minimum using this type of examination with the assurance that smaller lesions will be visible on the resulting radiographs than can be visualized fluoroscopically *(Fig. 7)*. Consequently, I feel that the double contrast technic should be mandatory in the examination of the colon of all children who have evidence of blood in the stools.

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DISCUSSION *(Abstract)*

Dr. A. Judson Graves, Jacksonville, Fla.—The technic of double contrast study of the colon has undergone evolutionary changes in the past several years; and like many other roentgen examinations it has improved, is technically less difficult than when first conceived and worked out, and, as a diagnostic maneuver, it is now more accurate than it was some years ago.

In my own experience, I feel I do this type of examination better since reading Dr. Moreton's work, and personally discussing his technic with him.

I shall not be presumptive enough to try to add emphasis to the well-discussed points included in the paper, but in my own reports to referring physicians, I emphasize the number and location of demonstrated polyps. I do not feel as a radiologist that I can take a dogmatic stand that other polyps are not present, either near or distant to the ones I am able to demonstrate on one specific x-ray examination. In the end, their removal is the responsibility of the surgeon. It behooves the surgeon to search for additional polyps even if it means using a scope through incisions in the colon at the time of operation.

I wish to ask Dr. Yates: Why do you use three enemas on the morning of the examination? How much water is used for each enema? Do you experience difficulty from fluid's remaining in the colon at times and preventing a satisfactory examination?

Dr. Robert D. Moreton, Fort Worth, Tex.—Dr. Yates has a fine collection of polyps in children. I have seen many of the studies which he has made.

He has well emphasized the problems of preparation which are the same as those noted in adults. He has slightly modified the routine type of air procedures to make them more adaptable to these small children.

Dr. Yates (closing).—I have had the pleasure of working with Dr. Moreton several years and he has taught me most of the radiology that I know.

With reference to Dr. Graves' questions, I do not know why we use three enemas instead of two or four. In studying preparation on several thousand colons we thought castor oil the best cathartic but, according to the pharmacopeia, castor oil acts only on the small bowel and has no action on the colon. We tried using no enemas but had to stop that practice almost immediately due to the large amount of retained material in the colon. Apparently the enemas stimulate contraction of the colon sufficiently to cause it to empty itself. I do not like for my patients to use more than one pint of solution with the enema. Larger amounts seem to leave solution in the right colon which interferes with adherence of the medium to the wall of the colon. This is especially true of elderly patients with an atonic colon. I do double contrast studies on some patients with colostomies and I have yet to see one with a clean colon regardless of the type of preparation used.

With regard to the bleeding, practically all of it appears to be bright red blood, although I may have seen dark blood in one or two. The 100 cases I mentioned were quoted from the series of Dr. Jack Kerr of Dallas, Texas.

I should like to sound a warning here. In doing colon examinations, one cannot be complacent and depend upon routine views for all cases. A group on the West Coast use upright and lateral decubitus films routinely, I think, and from what I have seen, are doing a good job. I would not begrudge anyone the use of films in any position he finds useful. In my own practice I have not seen upright and decubitus films that would compare in quality with the usual horizontal films. In the original description of the double contrast technic, Fisher in 1925 really covered the subject. He said that the colon could be partially or completely filled with medium; it could be partially or completely emptied or not emptied at all before the air was introduced. Roentgenograms could then be made in prone, suppine, upright or any other positions deemed advisable.

THE VALUE AND USES OF GASTROSCOPY*

By TIM J. MANSON, M. D.
Chattanooga, Tennessee

Since 1932, when Schindler[1] invented the modern flexible gastroscope, and the following year, when Benedict began using it in this country, gastroscopy has steadily gained in popularity, as more and more clinicians are recognizing its value. As with many other diagnostic procedures, it is not perfect, and consequently has had, and still has, its detractors.

It is true that there are "blind spots" of the stomach as seen through the gastroscope. And yet, as gastroscopists become more familiar with their instruments, and acquire greater skill in their use, and in simultaneous palpation of the stomach, these blind spots can be minimized, or in some instances, eliminated. The newer types of gastroscopes, with controlled flexibility of the tip, overcome many of these purely mechanical problems. Benedict[2] has recently described the

*Read in Section on Gastroenterology, Southern Medical Association, Forty-Fifth Annual Meeting, Dallas, Texas, November 5-8, 1951.

operating gastroscope, which may prove to be another forward step. In this instrument there is an extra channel through which passes a biopsy forceps, with which, under direct observation, one can obtain tissue for microscopic examination. Several objections have been raised to this procedure; the most valid ones are that hemorrhage may occur, undetected, after the instrument is withdrawn, and that, in the differentiation between benign and malignant ulcerations of the stomach, the tissue removed might be from an uninvolved area of the lesion, even though cancer might exist in another section.

FIG. 1

Case 1. No lesion apparent on films. Antral rigidity noted at fluoroscopy.

FIG. 2

Case 1. Drawing of normal prepyloric area as seen through gastroscope.

In what fields lies the importance of gastroscopy to the gastroenterological internist, or, for that matter, to the surgeon, or other practitioners?

Of referred cases, the most common problem presented to me is that posed by a gastric lesion, demonstrable by x-ray, about which there is doubt as to whether it is benign or malignant. One must emphasize here that there is no longer any question of which is the better, x-ray or gastroscopic examination of the stomach. These procedures should supplement, rather than compete with each other, and much greater diagnostic accuracy can be obtained by the joint use of both, than by the use of either one alone. It is well known that there will be a small group of cases in which either the radiologist or the gastroscopist can visualize lesions which cannot be seen by the other method. What is more helpful, however, is the agreement or difference of opinion held by the two examiners. Neither one is going to be right all of the time, and a selection of the proper method of treatment must depend upon a correlation of all the available tests, as well as the x-ray and gastroscopic findings. On the basis of comparison in a series of his cases, Benedict[3] has pointed out that,

> "If the gastroscopist can get a satisfactory view of the lesion, his chances of arriving at a correct diagnosis appear to be better than those of the radiologist."

The uninitiated often marvels at the seeming ease with which the gastroscopist can correctly call a gastric ulceration either benign or malignant, but to the trained eye, these lesions, when typical, have very different appearances. The ragged, nodular, infiltrating edge of the carcinoma, and its dirty gray floor, are usually distinctly different from the sharply punched out benign ulcer. It is still controversial as to whether these two conditions are separate entities, or whether some benign ulcerations do degenerate into malignant ones, or whether all such lesions must be considered as malignant and be resected. Most gastroscopists, however, feel that these are separate diseases, and as yet are not prepared to follow those who advo-

cate radical surgery in all ulcerative lesions of the stomach. There are inevitably going to be some instances, however, where there is doubt in the minds of both the radiologist and the gastroscopist as to the malignancy or innocence of an ulcer. In such cases one can only weigh all of the evidence, and, I believe, advocate surgery unless one is sure the lesion is benign. If any number of gastric carcinomas are to be cured, one cannot afford to wait until a questionable lesion is definitely a malignant one before deciding upon surgery. If, under such circumstances, one has a patient operated upon, as it turns out unnecessarily, he need not be ashamed of his decision. On the other hand, there will be numerous cases wherein one is so sure that a given ulcer is benign that medical treatment can be readily decided upon. By all means, however, these cases should be subjected to repeated x-ray and gastroscopic examinations until the risk of cancer has been eliminated.

Although carcinoma is the most pressing problem to most clinicians, one must mention the other important gastric lesions whose identification is made possible by gastroscopic observation. These include polyps, leiomyosarcomata, lymphosarcomata, and other such tumors. After one has had the satisfaction of viewing such lesions, at times previously undiagnosed, he is apt to agree with Chester Jones,[4] who said:

"The demonstration of a gastric lesion by the radiologist should usually imply confirmation by the gastroscopist."

Another field in which gastroscopy is extremely important is in the otherwise unexplained case of upper gastrointestinal hemorrhage. One is no longer justified in accepting an x-ray diagnosis of a normal stomach in such instances, where gastroscopy is available. Polyps or other tumors may be too small or inaccessible to be seen by x-ray and yet be the source of gross bleeding. The same may be true of an ulceration. It has been amply confirmed that gastritis[5] often is the cause of bleeding and gastroscopy is the only method by which this very real disease can be diagnosed. Although it is often suggested by the findings at x-ray, more often than not this is not borne out by gastroscopy, and many stomachs which appear normal radiologically, may show large areas of gastritis with active bleeding points.

We are all too often worried by the patient whose x-rays are entirely negative, and yet who continues to have various digestive complaints. I feel that in such cases examination cannot be considered complete without gas-

FIG. 3

Case 2. Film shows large irregular filling defect in antrum.

FIG. 4

Case 2. Gastroscopic view of lesion, proved to be lymphosarcoma.

troscopy. True enough, examination does not always reveal evidence of organic disease, for a majority of these people will turn out to have functional disorders, yet frequently one does find a gastritis, an undetected ulcer, early carcinoma or other lesion to be the explanation for their distress. Conversely, one must realize that in these doubtful cases, a negative gastroscopy can also be of tremendous help in clarification of the diagnostic problem.

A most valuable contribution can be made

FIG. 5

Case 3. Film showing suspicious filling defect high in the cardia.

FIG. 7

Case 4. X-ray showing inconstant, irregular defect at gastrojejunal stoma.

FIG. 6

Case 3. Drawing of lesion as demonstrated at gastroscopy. Carcinoma of stomach.

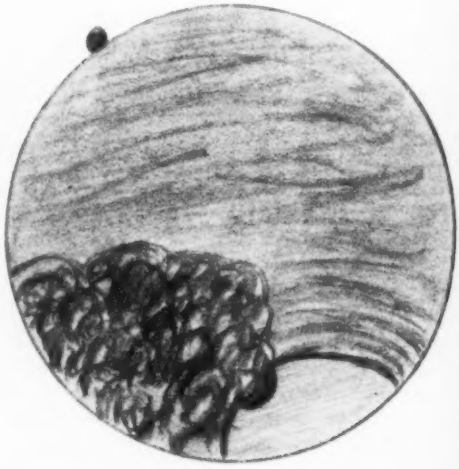

FIG. 8

Case 4. Recurrent carcinoma of stomach, as seen at gastroscopy.

by the gastroscopist in patients who have previously undergone gastric surgery, without good results. Effective x-ray studies may be difficult to make at times, whereas through the gastroscope the examiner may often demonstrate a poorly functioning stoma, gastritis, marked edema, or perhaps an ulcer at the stoma.

Case 1 (Fig. 1) is a group of x-ray films of the stomach of a 58-year-old man, which show no demonstrable pathology. At fluoroscopy, however, the radiologist felt that there was some unusual rigidity of the antrum, and suggested that the patient be gastroscoped.

Figure 2 is a drawing of a view of the prepyloric area, as seen at the gastroscopic examination. This was a perfectly normal stomach, and the "rigidity" that was felt to be suspicious at fluoroscopy could be interpreted as spasm, and carcinoma eliminated as a possible diagnosis.

Case 2 (Fig. 3), a 48-year-old woman, shows a large irregular antral filling defect, the extent and character of which were undetermined. A gastroscopic view is drawn in *Figure 4*. Here, just proximal to the angulus, could be seen a large, sharply defined mass protruding into the lumen of the stomach. The entire lesion was rounded, with a raised smooth, reddened border. In the center of this lesion was an irregular ulceration, with undermined ragged edges, and a dirty gray base. At the distal edge this ulceration seemed to extend into the surrounding border. We felt that this was a malignant tumor, but that its appearance was not that of the usual carcinoma. Gastric resection was performed, and the lesion proved to be lymphosarcoma. The patient subsequently died despite postoperative x-ray therapy.

Case 3 (Fig. 5) is one of a series of films made of a 53-year-old man. High in the cardia the radiologist felt there was a filling defect, but it could not be demonstrated constantly. The patient was gastroscoped and as the instrument was withdrawn, very high at depth III, one could see a grayish, irregularly nodular tumor mass occupying the entire left half of the field, toward the lesser curvature and anterior wall. This is illustrated in *Figure 6*. A total gastric resection was done, but the patient died of pneumonia several months later. The pathologic diagnosis was carcinoma of the stomach.

Case 4 (Fig. 7) is that of an 82-year-old man, who had had a gastric resection for carcinoma of the stomach in 1947, a year before this study. He had had a return of mild epigastric post-prandial distress, and at this examination, the radiologist felt there was a probable filling defect just above the gastrojejunostomy. This was amply confirmed at

FIG. 9

Case 5. Apparently normal stomach of patient with hematemesis.

FIG. 10

Case 5. Drawing of polyp visualized in fundus upon gastroscopy. A smaller polyp was also found distally.

gastroscopy, when a large tumor mass could be seen above the stoma *(Fig. 8)*. Because of his age and poor general condition, further surgery was not employed and this patient subsequently died of metastatic carcinoma.

Case 5 illustrates one of the valuable contributions that gastroscopy can make in instances of gastric bleeding. This 52-year-old woman on several occasions had regurgitated bright red blood, never in large amounts.

FIG. 11

Case 6. Film of patient with hematemesis appears normal.

FIG. 12

Case 6. Small gastric ulcer demonstrated through gastroscope.

Alarmed, she had had three x-ray examinations by very competent radiologists, each of whom could find no evidence of organic disease. An apparently normal film is shown in *Figure 9*. Gastroscopy, however, showed two polyps, a peanut-sized one on the anterior wall, about 2 cm. proximal to the angulus, and a larger one, in the fundus, hanging down like a red grape, as shown in *Figure 10*. This patient was successfully operated upon and the two polyps were removed. Pathological study confirmed the gastroscopic impression that these were benign polyps.

And finally, *Case 6* is that of another patient with hematemesis, a 56-year-old man who had symptoms suggestive of an ulcer but with repeatedly negative gastrointestinal x-rays, as shown in *Figure 11*. At gastroscopy, with the instrument at depth I on the posterior wall, about 1 cm. distal to the angulus, one could see a very shallow, punched out ulcer with sharp, smooth edges, as illustrated in *Figure 12*.

It was felt that this lesion was benign, and since treatment, periodic re-examinations during the past 4 years have shown it to be healed.

These cases, obviously weighted in favor of the gastroscopist, should not be construed as an attempt to discredit radiology. Similarly, cases could be presented to show lesions, demonstrable by x-ray, yet not seen by the gastroscopist. Rather we must take from the comparison one lesson. Gastroscopy can be of tremendous value to us in our diagnostic problems, and in doubtful instances should be thought of and used more frequently than it is at present.

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DISCUSSION *(Abstract)*

Dr. John S. Atwater, Atlanta, Ga.—The conventional procedure of gastroscopy is usually adequate, yet has certain distinct disadvantages. To some patients the examination has been most unpleasant in

spite of the fact that the gastroscopist has attempted to prepare his patient psychologically prior to the examination. A medical student told me, after undergoing gastroscopy in the hands of one of our most eminent gastroscopists, that he felt as though he were being held down on the examining table by a hot rod thrust from his mouth, through his chest, into his abdomen. Certainly, excitable patients object to this procedure even more than did this relatively stable young medical student. Oftentimes patients have tended to gag and to move about on the examining table limiting the time available for good gastroscopic observation. From a teaching standpoint, the time allowed the examiner to show students just what he is looking at, to describe the lesion, and for the students, in turn, to identify the lesion in question was often exceedingly limited.

Early in 1947 we decided to try alternate patients on local or general anesthestic approaches for gastroscopy. Invariably, the patients who experienced general anesthesia viewed the examination as very satisfactory. On the other hand, many in whom local agents were used, expressed the hope that they need never have gastroscopy in the future. Such an expression is not peculiar to this observer for I have heard patients of other gastroscopists express similar attitudes following gastroscopy under local anesthesia.

In our earlier examinations we used pentothal sodium,® which was quite satisfactory. However, upon the introduction of surital sodium,® we found that the recovery period was so much more rapid that we have changed to the exclusive use of surital sodium.® In conjunction with the pentothal® or surital® we have used curare intravenously and oxygen intranasally.

In well over a hundred gastroscopies with such an approach, we have had no postoperative complications. As a rule, the patient could be awakened before leaving the operating room and with few exceptions, the remaining patients have awakened soon after reaching the recovery rooms. Nourishment and early ambulation has been encouraged. Usually the patient could be discharged from the hospital on the same day he was admitted avoiding any additional expense or taking up a muchly needed hospital bed.

From the gastroscopist's point of view the examination under this technic is ideal in that time is not an important factor. There is no longer the need for quick glances at important areas because of discomfort to the patient or the changing local picture. Lesions can be identified to multiple observers and hence this method affords an excellent teaching medium. When desirable the patient's position may be changed to the prone or the right lateral position for additional views of the interior of the stomach without inconvenience to him. While the taking of a biopsy by using the Benedict operating gastroscope may not be difficult, yet anyone who has performed such a procedure realizes that at times it may be tedious. In the locally anesthetized patient, the biopsy procedure may prolong the examination and add greatly to the unpleasantness of it for the patient. With intravenous anesthesia, the examination may still be prolonged but the unpleasantness is gone and, of course, multiple biopsies may be taken with no additional risk or discomfort to the patient.

Dr. Ernest Leonard Posey, Jr., Jackson, Miss.—Investigation of the roentgenologically deformed antrum, study of indeterminate gastric lesions, differentiating benign and malignant gastric ulcers, and more effectively following the healing process in benign ulceration are prime indications for gastroscopy as we have been shown.

Gastroscopy would be a valuable procedure even if limited entirely to instances of unexplained upper gastrointestinal hemorrhage. In this unique category the gastroscope will disclose hemorrhagic gastritis in a surprisingly large percentage of cases, in my experience far outnumbering the various other gastric lesions. The diagnosis is established only by endoscopy. Proper treatment may not be administered or useless surgery may be performed should the gastroscope not be utilized in this group.

There is now no question that emotional tension may be reflected by visible changes in the gastric mucosa. These alterations can easily be studied via the gastroscope. A gastroscopic diagnosis of psychoneurosis is in my opinion completely justified. The red rugae of a gastric neurosis are the equivalent of the blushing bowel seen proctoscopically in mucous colitis. Gastroscopy therefore has a place in psychosomatic medicine.

The peculiar mucosal atrophy associated with pernicious anemia is considered a potential precursor of carcinoma. Gastric malignancy is estimated to occur in as high as 15-20 per cent of these patients. Early diagnosis is essential. A yearly gastroscopic examination is recommended in every case of addisonian anemia.

Gastroscopy is a valuable procedure but proportionately no more so than any other investigational method. It is tempting for the gastroscopist to become over enthusiastic concerning his art. However, the gastroscopist is also a clinician and as a clinician realizes that in properly selected cases his instrument provides a means toward an end and is no end in itself.

VERTICAL FLAP URETEROPELVIOPLASTY*

PRELIMINARY REPORT

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Obstruction at the ureteropelvic juncture can be corrected by any one of a number of surgical procedures. However, the success of these operative alterations is not uniform and over the years has provided a fertile field for

*Read in Section on Urology, Southern Medical Association, Forty-Sixth Annual Meeting, Miami, Florida, November 10-13, 1952.

surgical research. No single surgical procedure has proved to be a panacea for the many variations and anomalies associated with the ureteropelvic area and it has been perhaps the attempt to fit a single procedure to the variations in pathology that has forced one method after another to fall into disuse.

Methods designed to overcome ureteropelvic obstruction culminated in 1937 in the "Y-V plasty." Following a rather exhaustive research and the application of his procedure, Foley presented his favorable results.[1] However, this excellent method found its greatest application in cases of stenosis and achalasia associated with high ureteral insertion. When applied to the dependent type of ureteropel-

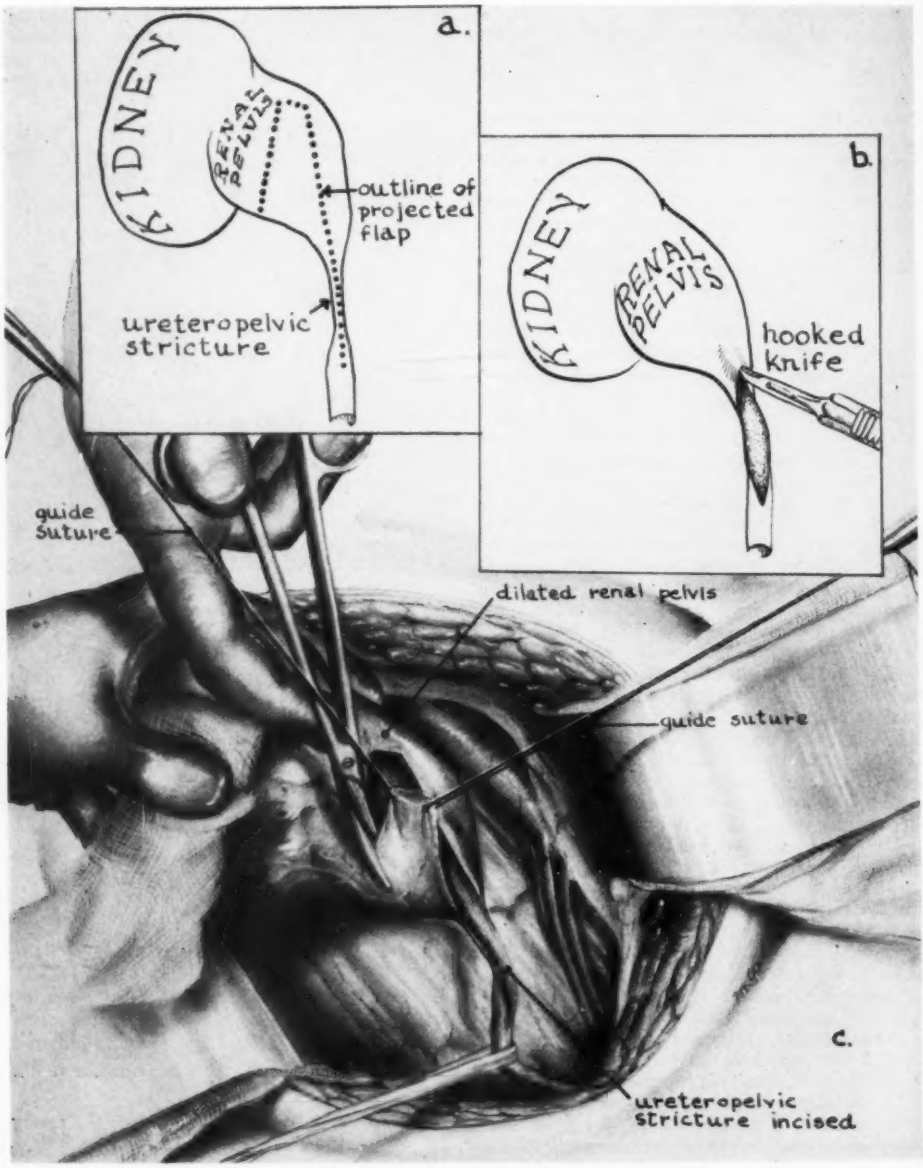

FIG. 1, a, b, and c

vic juncture obstruction, a change in the vertical axis of the kidney occurred or a tissue redundancy was produced. It was apparently this type of obstruction as well as long strictured areas of the ureter which prompted Davis and his colleagues[2] to introduce their method of intubated ureterotomy. When applied as outlined by the authors, the above methods of ureteroplasty produced encouraging but not uniformly satisfactory results. Recently, Culp and DeWerrd[3] have reported their observations of a method of pelvic flap for correction of certain types of ureteropelvic obstruction.

During an operation on September 5, 1951 on a 24-year-old white woman whose diagnostic preoperative work-up revealed a narrowed ureter for 2 cm. below a dependent ureteropel-

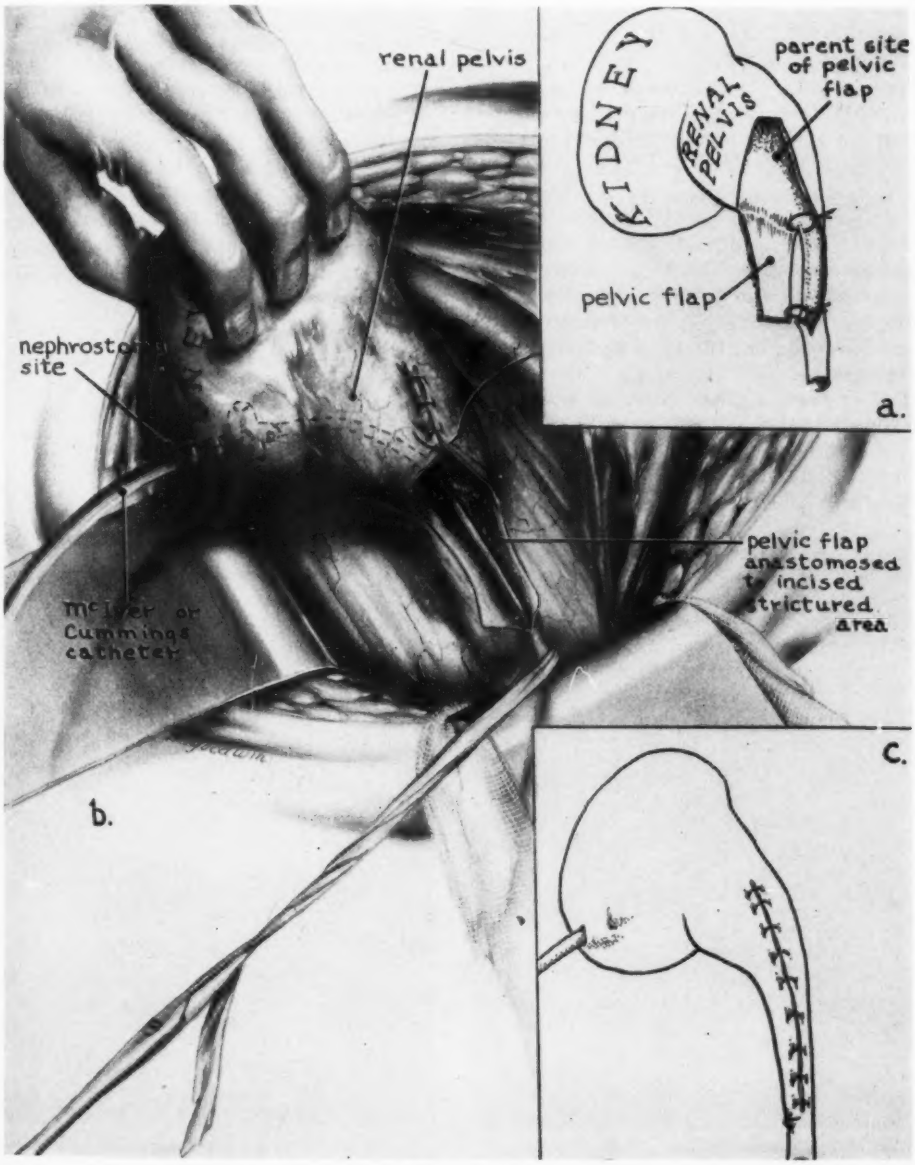

FIG. 2, a, b and c

vic juncture, a method of correction gradually evolved.

Operation.—The required length of pelvic tissue is determined by the extent of the ureteral obstruction *(Fig. 1a.)* The ureteral incision is begun distal to the obstruction and carried proximally into the redundant pelvis *(Figs. 1b and 1c)* to a point previously determined; then carried laterally for approximately 2 cm. and distally to the ventrum of the pelvis, the incision paralleling the initial pelvic incision *(Fig. 1c).* The flap is anastomosed to the incised strictured area with interrupted sutures of 0000 chromic catgut *(Figs. 2a and 2b).* A McIver or Cummings' catheter is introduced via a lower pole nephrotomy into the ureter and pelvis *(Fig. 2b).* The remaining pelvis and ureteral incisions are closed with interrupted sutures of 0000 catgut *(Figs. 2b and 2c).* The approximation of the pelvic edges automatically reduces the lumen of the redundant pelvis. However, if necessary further reduction in pelvic size can be accomplished by excision of additional redundant pelvic tissue.

Twelve cases of ureteropelvic obstruction of the "dependent ureter" type have undergone vertical flap ureteropelvioplasty, since September 1951. Six of the cases are reported in detail.

CASE REPORTS

Case 1, Fig. 3A.—Preoperative right retrograde pyelogram. Right hydronephrosis, grade 3. *Fig. 3B.*—Postoperative right retrograde pyelogram (14 months). Reduction in hydronephrosis, persistent wide funnel-shaped ureteropelvic juncture.

A married woman aged 24 was seen in September 1951. Her first episode of urinary tract infection occurred in 1938. There was occasional right flank discomfort. She had right hydronephrosis grade 3, and a normal left kidney. At operation the right pelvis was tense and dilated. The ureteropelvic junction was obstructed by a dense stricture for 2 cm. The latter was incised and a vertical flap was brought down alongside. The pelvis was reduced in size. Nephrostomy and nephropexy were performed. One year follow-up reveals reduction in hydronephrosis. The urine is sterile.

This is the initial case in which the operative technic was applied. A young married woman with advanced hydronephrosis would ordinarily be a candidate for nephrectomy, but application of the procedure appeared to be justifiable in view of age and marital status. After 14 months, the patient has required no medication, has been asymptomatic and, while it is early for final decision, perhaps the procedure was warranted at her age.

Case 2, Fig. 4A.—Right retrograde pyelograms. Hydronephrosis grade 3-4. Ureteropelvic junction obstruction. *Fig. 4B.*—Postoperative (14 months) right retrograde pyelogram. Marked reduction in hydronephrosis. Persistent funnel-shaped ureteropelvic junction.

FIG. 3A, CASE 1

FIG. 3B, CASE 1

In September 1951, a 23-year-old married man was seen. He gave a three-year history of right flank pain with associated episodes of recurrent urinary tract infection. He was acutely ill for 3 weeks with nausea, vomiting, chills, and fever, and an exquisitely tender right flank. He had right hydronephrosis grade 3-4. At operation, the right renal pelvis was dilated and tense. An extrinsic obstruction due to renal fascia constricted the ureter at the ureteropelvic junction. A vertical flap ureteropelvioplasty was done, and a nephropexy. Function is normal. The urine is sterile, and he is asymptomatic.

A young male whose right renal obstruction was due to extrinsic fascial obstruction might have had as good a result merely by freeing the ureter from the surrounding fascial tissue. However, application of the surgical procedure appeared to be in order and from the result was justifiable. At least, no stricture has occurred at the site of the corrective operation.

Case 3.—In September 1951, a 33-year-old married woman was seen. For 10 years she had had intermittent episodes of right costovertebral angle pain, chills, fever, urinary frequency, dysuria, and weight loss. She had a tender right flank, palpable kidney, and right hydronephrosis, grade 2-3. The ureter was constricted for 3 cm. at the ureteropelvic juncture. The pelvis was dilated and tense. Vertical flap ureteropelvioplasty and nephropexy were performed. Function improved. Hydronephrosis was reduced. The ureter was dependent and the urine sterile. The patient is asymptomatic relative to right kidney.

The hydronephrosis was not great. The renal cortex was excellent. The obstruction was due to an intrinsic abnormality. Application of the principle described resulted in a good early result, but the ureter and pelvis are not in so desirable a position as should have been obtained.

Case 4, Fig. 5A.—Right retrograde pyelogram. Grade 3 hydronephrosis. Ureteropelvic junction obstruction. *Fig. 5B.* Ten months postoperative retrograde pyelograms. Appreciable improvement in hydronephrosis. Wide funnel-shaped ureteropelvic junction.

In January 1952, a 28-year-old married woman was seen. She had had recurring right flank pain for 22 years associated with episodes of urinary tract infection. There was moderate tenderness in the right flank, and urinary tract infection. At operation the right kidney was found to be hydronephrotic with moderate destruction of the cortex. The pelvis was tense and dilated, the ureter narrow for 3 cm. at the ureteropelvic junction. Vertical flap pelvioplasty was done. She is asymptomatic and the urine is sterile.

A young married woman whose renal destruction was moderately far advanced would under usual circumstances warrant nephrectomy. However, the surgical procedure was applied with success and after 10 months improvement has been maintained.

Case 5.—In February 1952, a 15-year-old girl was seen, with constant right flank pain and nausea. She had urinary tract infection. At operation the renal cortex was seen to be excellent. The pelvis was dilated

FIG. 4A, CASE 2

FIG. 4B, CASE 9

moderately. Observation of ureteral peristalsis revealed obstruction at the ureteropelvic junction. Vertical flap pelvioplasty was performed. The urinary tract infection cleared. Flank discomfort was less severe. Renal function was good. There was a decrease in renal dilatation.

Whether the degree of obstruction warranted surgical intervention is debatable in this case. However, in view of the pain and persistent infection of the right kidney and the age of the patient, it was felt that rather than await progressive hydronephrosis, surgical intervention was justifiable. Little obstruction was observed at surgery. It is difficult both objectively and subjectively to determine how much improvement has occurred. However, on a research project one point has been demonstrated. With the procedure there has been no evidence of stricture formation at the site of repair at this early date.

Case 6.—In April 1952, a 22-year-old married woman was seen with a 2-year history of right hip and right flank pain, right lower quadrant. She had a urinary tract infection, and right hydronephrosis grade 4. At operation the right kidney showed advanced hydronephrosis. The pelvis was dilated. An aberrant artery at the ureteropelvic junction supplied the remaining lower pole kidney. Vertical flap ureteropelvioplasty was performed. Pain was relieved. Function was poor. There was no appreciable change in hydronephrosis. The ureteropelvic junction was patent.

Nephrectomy should have been done in this case. However, seven months following the procedure, the patient has been asymptomatic, the pelvis and ureteropelvic junction remain funnel-shaped, and the kidney empties, but renal parenchymal damage was so great that we would expect little improvement in function. An opportunity to observe the results of the procedure in such far advanced destruction presented itself and was taken.

Six additional cases have undergone vertical flap ureteropelvioplasty, but it seemed premature to report them in detail at this time. Of this group, the ages ranged from 12 to 35 years. The symptoms, physical examination, laboratory findings, and roentgenologic examinations were similar to the cases in the first group of six. The results have been encouraging.

ANALYSIS OF CASES

Of the twelve cases subjected to vertical flap ureteropelvioplasty, results can be considered only as early observations and not long range conclusions. The oldest cases are now more than 14 months since operation. The youngest are 3-6 months old. In all cases the operative procedure was the same. The nephrostomy catheter was removed five

FIG. 5A, CASE 4

FIG. 5B, CASE 4

weeks postoperatively in all except *Case 2*, in which the catheter was removed in two weeks and it was interesting to note that at this early date the result is encouraging. Three of the cases, those in which renal parenchymal destruction was far advanced, probably warranted nephrectomy. However, since this was an experimental study, application of the principle of necessity had to be done on all patients with the dependent type of ureteropelvic obstruction to determine the merits of the procedure under all conditions. Infection was present in the majority of cases at the time of surgery. Pre- and postoperatively, all patients received indicated antibiotic therapy depending upon urinalysis and cultures. None of the patients has required secondary surgery, although it is too early to predict what the eventual long range requirements will be.

CONCLUSIONS

Twelve cases of ureteropelvic junction obstruction have undergone vertical flap ureteropelvioplasty. Six of the twelve cases have been reported in detail. The results are encouraging. The investigation will be continued. Long range results will be reported later.

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DISCUSSION *(Abstract)*

Dr. Major F. Fowler, Atlanta, Ga.—All of us have long felt that our surgical procedures for dealing with obstructive lesions at the ureteropelvic juncture were not entirely adequate. This new procedure certainly appears to be sound surgically and from the results observed, is a superior method for dealing with the dependent type. The pelvic flap preserves the blood supply and offers unlimited width and length to reconstruct the narrowed ureter without disturbing the anatomical relations. We agree that nephrostomy and ureteral splinting, with the McIver or Cummings' catheter, afford greater stability and greater chance of a good result.

We have usually employed the Y-V plasty for the cases of high insertion of the ureter and resected the redundant pelvis. For the cases of intrarenal hydronephrosis and those with long strictured portions of ureter we have employed a modified Davis intubated ureterotomy. We have employed nephrostomy and splinting from above, using either a McIver nephrostomy tube or a malecot catheter and a red rubber French catheter of suitable size. Davis' method of splinting from below by means of a ureteral catheter has not been too successful in our hands because of the great difficulty in keeping it in position and since it allows less mobility of the patient.

Doubtless, we shall find ourselves trying to select the operative procedure best suited for the particular case and, in time, will probably employ all of the well established procedures. It has just been observed in the cases of dependent ureteropelvic juncture obstruction we could employ the vertical flap ureteropelvioplasty and have two chances of success provided the splinting catheter is left in place long enough. Even if the distal portion of the flap sloughed, healing of the ureter by granulation according to the principle of Davis' method would still take place.

Dr. J. C. Kimbrough, Washington, D. C.—In 1934 at a meeting of the American Urological Association, I presented a paper on the operative treatment of hydronephrosis. I had about a dozen cases, and I was very enthusiastic in those days. That has been a long time ago, and my enthusiasm was greater then than it is now. I tried all the good procedures. The further I went the worse the conditions became and I have not written anything about the operative treatment of hydronephrosis since then.

Dr. S. Joseph Pearlman, Miami Beach, Fla.—The subject of uretero-pyeloplasty is fascinating and efforts to correct obstructive uropathies at the uretero-pelvic junction and upper ureter will continue to tax the skill and ingenuity of the urologist. I am particularly concerned with strictures in the upper ureter where flaps are not possible because of intra-renal pelves. Cutting the ureter at the site of constriction with a side-to-side anastomosis has been suggested, but I believe that this type of procedure will ultimately result in a hydro-ureter and hydro-nephrosis, because of injury to the sympathetic nerve supply. In suturing the pelvis and ureter most urologists shy away from the use of through and through sutures and content themselves with sutures approximating the adventitial coats only.

With regard to the use of a splint catheter, it seems to me that in the Davis type of ureteral intubation the catheter brought down the ureter into the bladder should remain *in situ* for at least 6 to 8 weeks.

In the pyeloplasties done routinely, the splint catheter can safely be removed after 4 to 6 days. Strictures in the ureter, secondary to the use of splint catheters, can be prevented, if we follow the Davis method of bringing the catheters well into the bladder.

It is important, too, that the catheters fit the ureteral lumen snugly, but not too tightly.

Dr. Scardino (closing).—It is encouraging to learn that the type of procedure presented, or similar technics, have been used successfully by the discussors. I found the vertical flap technic promising in cases of ureteropelvic obstruction of the dependent type but conclusions were reserved for the long term follow-up. We can perhaps anticipate, from the experiences of the discussors, good long term results.

TEN YEARS EXPERIENCE IN SURGICAL MANAGEMENT OF CANCER OF THE LUNGS*

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and
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In the last ten years we have had the opportunity to examine one hundred patients who had a diagnosis of cancer of the lung, in 77 of which it was based on biopsy or smear. Only six of them were rejected for surgery on the basis of metastasis to supraclavicular lymph nodes or other organs.

A thoracotomy was performed on 94 patients in 22 of whom a resection was not possible. Of those who had an exeresis 69 had total pneumonectomy and three lobectomy.

Lobectomy was carried out as a curative measure in two cases. One of these patients died two years later from ipsolateral metastasis. The operation was carried out as a palliative measure in another one whose respiratory function was impaired by emphysema.

Histologic examination of five resected specimens, four pneumonectomies and one lobectomy proved that the preoperative diagnosis of cancer was wrong, the lesions being of tuberculous origin.

We had 94 males and 6 females, 89 being white and 11 colored. Fifty-seven had the tumor in the left side, forty-three in the right. The oldest case was 68, the youngest eleven, the mean, age 48.

Our operative mortality is 5 per cent and our hospital mortality 6 per cent.

The average time elapsed from the beginning of the development of symptoms to operation was 5½ months.

Of the 77 cases in which microscopic study was done, 49 were squamous cell carcinoma, 25 adenocarcinoma, 2 oat cell carcinoma and one alveolar cell carcinoma.

Of the 69 pneumonectomies, only 18 were free from lymph node involvement in the mediastinum and it should be emphasized that these are the cases which have yielded the best survival rate which up to now is as follows:

Survival Time, Years	No. Cases
8	1
6	1
4	15
3	19
2	14
1	10
0.5	4

Adenocarcinoma prevailed in peripheral lesions, while squamous cell carcinoma was more often found in centrally situated tumors.

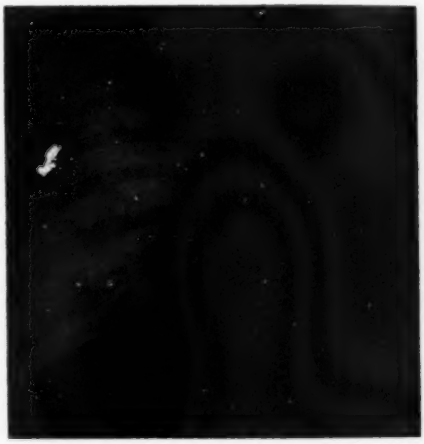

Fig. 1

Case 1, Preoperative.

Fig. 2

Case 1, Postoperative.

*Read in Section on Surgery, Southern Medical Association, Forty-Sixth Annual Meeting, Miami, Florida, November 10-13, 1952.

In spite of the fact that adenocarcinoma has a marked tendency to produce metastasis, our two patients alive after 6 and 8 years had this kind of tumor. All but one of the 15 alive after four years had squamous cell carcinoma.

Although it is obvious that in a disease that treated otherwise would produce a 100 per cent mortality, a single surgical cure would recommend the procedure, the experience derived from our short series is not presented with the purpose of bolstering the surgical treatment of cancer of the lung but to emphasize once more the great value of exploratory thoracotomy.

Prompt use of exploratory thoracotomy is responsible for the excellent result achieved in the following cases.

Case 1.—B. G., a white man forty years old complained chiefly of bloody sputum. His personal history was non-contributory. In the family history, tuberculosis was noted. X-ray showed atelectasis. Bronchoscopy revealed a polypoid tumor of the left main stem bronchus. Microscopic study showed adenocarcinoma. The duration of symptoms before operation was 1 month. Operation was left pneumonectomy.

Case 2.—V. T., an 11-year-old white boy, complained of fever, cough, and dyspnea. Personal and family history were non-contributory. X-ray revealed atelec-

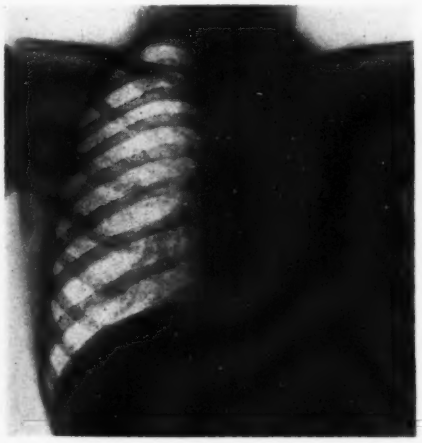

FIG. 3

Case 2, Preoperative.

FIG. 4

Case 2, Postoperative.

FIG. 5

Case 3, Preoperative.

FIG. 6

Case 3, Postoperative.

tasis of the left lung. Bronchoscopy showed a polypoid tumor of the left main stem bronchus. Biopsy demonstrated bronchial adenoma (adenocarcinoma grade I?). Duration of symptoms before operation was 4 months. Operation was left pneumonectomy.

Case 3.—A. P., 56 years old, a white man, complained chiefly of cough. Personal and family history were non-contributory. X-ray showed a small round area of increased density in the left upper lobe. Bronchoscopy was irrelevant. Duration of symptoms before operation was 25 days. Thoracotomy showed tumor in the left upper lobe. Frozen section showed epidermoid carcinoma. Operation was left pneumonectomy.

Exploratory thoracotomy cleared up the diagnosis and prognosis in other cases.

Case 4.—A. N., 46 years old, a white man, complained chiefly of bloody sputum. Personal and family history were non-contributory. X-ray showed atelectasis of the lingular lobe. Bronchoscopy was irrelevant. Duration of symptoms before operation was 2 months. At thoracotomy induration of the lingular lobe was seen. Frozen section showed chronic pneumonitis. At operation, segmental resection was done.

Case 5.—F. M., 42 years old, a white woman, complained chiefly of pain and fever. Personal and family history were non-contributory. X-ray showed a round area of increased density in the left infraclavicular region. Bronchoscopy was irrelevant. By selective bronchography, a bronchial stop was located at the posterior branch of the left upper lobe. The duration of symptoms before operation was 5 months. Thoracotomy showed a tumor in the left upper lobe. Operation was left pneumonectomy. Postoperative microscopic study revealed a tuberculoma.

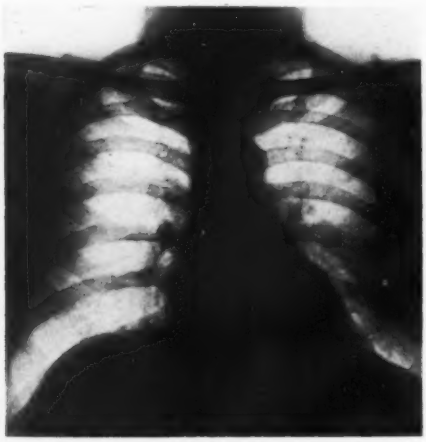

FIG. 7

Case 4, Preoperative.

FIG. 8

Case 4, Postoperative.

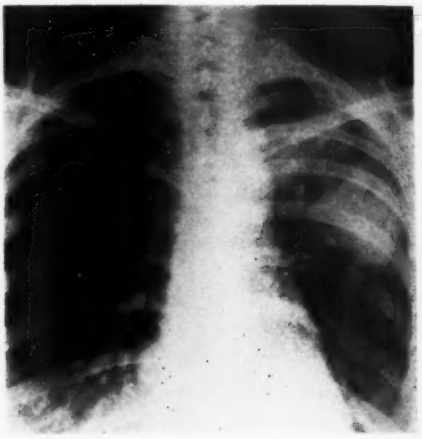

FIG. 9

Case 5, Preoperative.

FIG. 10

Case 5, Postoperative.

Exploratory thoracotomy proved of great value in cases like the following, which seemed inoperable. None the less the tumor was resected and the patient is still alive 4 years after the operative eradication.

Case 6.—A. F., 62 years old, a white man, complained of pain and fever. Personal and family history were non-contributory. X-ray showed increased density in the left infraclavicular area. A tumoral mass was demonstrated. Bronchoscopy was irrelevant. Duration of symptoms before operation was 7 months. Thoracotomy showed a cancer of the left apical segment with invasion of the ribs. Operation consisted of left pneumonectomy and block resection of the infiltrated ribs.

SUMMARY

Reviewing the statistical series reported by leading surgeons of the United States, we find the following resectability rates:

Year	Author	Per Cent	Total Cases
1940	Overholt	14	100
	Churchill	17.4	155
1947	Rienhoff	31	502
1948	Ochsner and deBakey	35	548

These rates are in sharp contrast with our 71 per cent resectability rate and we think it can be explained on the basis that our series started in 1942, at a time when the general practitioner had been alerted by countless papers on the early diagnosis and surgical treatment of cancer of the lung and thoracic surgery had attained a great development.

Survival rates compare favorably with results obtained from the surgical treatment of cancer of other organs.

As has been pointed out repeatedly in hundreds of papers dealing with this subject early diagnosis and early thoracotomy are the difference between success and failure in the surgical treatment of cancer of the lung.

FIG. 11

Case 6, Preoperative.

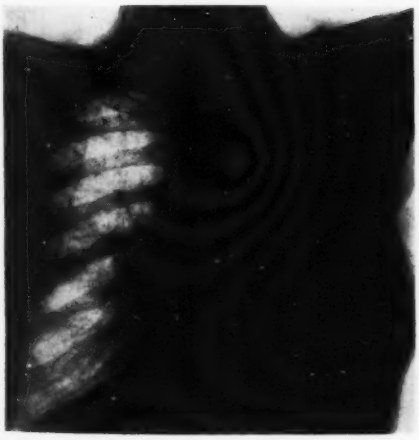

FIG. 12

Case 6, Postoperative.

DISCUSSION *(Abstract)*

Dr. Paul T. DeCamp, New Orleans, La.—I should like to suggest that the high resectability rate in the authors' series may, in part, be due to a difference in case selection. If I understand correctly, this series represents cases which have been referred to a surgeon for operation. It has been the custom in many of the North American clinics, certainly in New Orleans, to study and report all the patients with bronchogenic carcinoma admitted to a hospital or clinic whether surgical therapy was considered or not.

With respect to the duration of symptoms from onset to operation, the authors' experience has certainly been more favorable than ours in New Orleans. In a series of 826 unselected cases of carcinoma of the lung seen at the Ochsner Clinic or on the Tulane Service at the Charity Hospital in New Orleans the average delay from onset of symptoms to the application of definitive therapy has been 8.7 months. This period of delay has decreased slightly from an average of 10 months prior to 1942 to an average of 8 months during the past 5 years.

Unexpectedly, the resected cases gave a longer history, 10.5 months, than the non-resected cases, 7.5 months. This indicates that the resectable lesions represent a favorable slow-growing segment of the cancer population.

In spite of a lower resectability rate the Charity patients gave a shorter average history, 7.5 months, than the private patients, 9.3 months. We believe that this is due to the fact that the charity economic group either do not recognize early symptoms so readily, or do not reproduce a history so accurately.

It was possible to determine the patient and physician responsibility for delay in 707 cases, and it was found that the doctor was responsible for twice as

much delay as the patient, the average delay being 5.5 months and 2.8 months respectively. A most disturbing finding was that the physician handles his private patients particularly badly. The charity patient was responsible for an average delay of 3.0 months and his physician was responsible for an additional delay of 3.6 months. The average private patient delayed 2.6 months in seeing his physician, but the latter then caused an average additional delay of 6.2 months. This difference may be in part due to the difference in economic circumstances, and in part to the fact that the private patient seeks a physician's care earlier, but the fact still remains that there has been an average delay of over six months between the time the private patient first sees a physician and his subsequent referral for definitive diagnosis and therapy.

PRIMARY MENINGEAL MELANOMAS*

By SIMON RUSSI, M.D.†
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and
BENEDICT NAGLER, M.D.§
Richmond, Virginia

The first case of primary melanoma of the meninges was reported by Virchow in 1859.[1] Kessler[2] in 1937 reviewed 36 case reports of melanotic tumors which arose as primary meningeal growths and added 2 cases of his own, bringing the total to 38. One of the lesions reported by Kessler was a circumscribed melanoblastosis of the pia which the author considered to be of a benign nature. The lesion in the second case, which he classified as a melanosarcoma, involved the leptomeninges of the base of the brain. Schnitker and Ayer[3] in 1938 reviewed a series of 30 cases and added a case report of their own. Five of the cases listed in the series collected by Schnitker and Ayer did not appear in Kessler's review which brings the total of cases listed by both reviews to 43. We were able to collect 19 more case reports from the literature[4-18] which would bring the total of reported cases to 62. There have been a few more case reports published, particularly in the foreign literature, which were not available to us.

A review of the 19 recent case reports reveals that the clinical picture depends largely on the location of the lesions. Preoperative and ante mortem diagnoses have run the gamut of neurology, brain and spinal cord tumor being the most common. The lesions were not invariably fatal, there being three case reports[7 15] of nodular pigmented meningiomatous growths followed by a survival period ranging from 2 to 8 years. There have been no recorded cases where a preoperative or ante mortem diagnosis of meningeal melanoma was made, although Cardona in 1947 quoted a reference to Bau Prusak and Mackiewicz who in 1930 claimed to have diagnosed a melanoma of the central nervous system by finding melanin pigment in the spinal fluid.

In 5 cases of the 19, visceral implants were described. The implants were found in the stomach in 3 cases;[10 13 14] liver in 2 cases;[10 13] lung in 2 cases.[13 14] The pleura, diaphragm, pancreas, gallbladder, adrenal, lymph nodes and skin were involved once each.[8 13 14 18]

Benign cutaneous nevi were described in 2 case reports[6 9] where no other melanomatous growths were found. In 9 cases the meningeal lesion was the sole focus of melanotic new growth.

We have encountered two cases of melanoma of the meninges which we are reporting because of their rare incidence and because of some unusual clinical and pathological features.

CASE 1

History and Physical Findings.—A thirty-five-year-old white man was admitted to the Veterans Administration Hospital, Richmond, Virginia with a four-month history of progressive illness which began with attacks of vomiting accompanied by nausea occurring about two hours after meals. The nausea disappeared, however, after a few days. He also complained of intermittent, dull, frontal headaches. About six weeks prior to his admission he had a convulsive episode lasting approximately two hours without tongue bite or loss of sphincter control. He had lost about 30 pounds of weight since the onset of his illness. The family history was non-contributory.

After his convulsive seizure he was hospitalized for about six weeks in another hospital. There, the initial examination revealed a thin, well developed, white man who was quite lethargic. When aroused,

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*Read in Section on Pathology, Southern Medical Association, Forty-Sixth Annual Meeting, Miami, Florida, November 10-13, 1952.

*Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are a result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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he appeared mildly confused and disoriented. There was moderate nuchal rigidity. The pupils were dilated but equal and reacted to light and accommodation. Fundoscopic examination was negative at that time. The deep reflexes were decreased but equal. The abdominal reflexes were absent. There was a positive Babinski and questionable Kernig sign bilaterally. Blood pressure, pulse and temperature were normal. While in that hospital, the patient continued to vomit two or three times a day but did not complain of nausea. On one occasion, projectile vomiting was observed. He continued to be lethargic and became increasingly confused and disoriented. Before his transfer to the Veterans Administration Hospital in Richmond, Virginia he became irritable and intensely restless. His blood pressure rose to 154/102 and his pulse rate gradually fell to 44 to 48 per minute.

A physical examination performed upon admission to the Veterans Administration Hospital in Richmond revealed the presence of nuchal rigidity, positive Kernig, hypoactive deep reflexes and absent abdominal reflexes. The patient was semi-comatose. After a few days, all the deep reflexes disappeared. Numerous spinal taps performed in this institution as well as in the institution where he was previously hospitalized revealed a spinal fluid pressure which varied between 240 and 600 mm. of water.

Laboratory Findings.—Red and white blood cell counts were generally within normal limits. On only one occasion was there a leukocytosis of 13,900 with 84 per cent neutrophils. The hematocrit was 53. Bleeding and clotting times, as well as the sedimentation rate, were normal. Blood sugar and nonprotein nitrogen were within normal limits. Serologic tests for syphilis were negative.

Spinal fluid sugar ranged between 66 and 76 mg. per 100 cc., and on only one occasion dropped to 35 mg. per 100 cc. Cell counts revealed two to twelve lymphocytes. The chlorides were within normal limits. The Pandy test was 4 plus. Total proteins ranged from 313 to 550 mg. The spinal fluid formed a pellicle upon standing. However, tubercle bacilli were not found by smears or cultures performed on several occasions. X-ray examination of his chest revealed normal findings. The skull films were negative.

Course in the Hospital.—The patient's condition steadily deteriorated. He became increasingly irrational and lethargic. Slight papilledema appeared in both fundi, especially marked in the left. He developed a weakness of his left grip and ataxia of the left upper and lower extremities. His temperature fluctuated from 98.6° F. to 100° F., rising to 101° F. on two occasions.

Trephine openings were made in the parietal regions. The dura was noted to be under pressure but of normal color and consistency. When the dura was opened the brain herniated through the defects. No lesions could be visualized. One hundred eighty cubic centimeters of fluid were removed from the right ventricle and air was injected in its place. After manipulation, some gas escaped into the basal cisterna and spinal subarachnoid space. X-ray films revealed generalized enlargement of the whole ventricular system. There was no evidence of displacement of the ventricular system, and none of the sulci were visualized. After the ventriculogram, the patient developed a left facial weakness and his papilledema increased bilaterally to the point of blindness with only light perception remaining. Daily spinal taps were done to relieve the pressure, and the initial manometric readings varied from 300 to 600 mm. of water. Although he received supportive treatment, including antibiotic medication, the patient became comatose on the thirty-first day of his hospitalization and died on the thirty-fourth day. The clinical impression was: "Chronic arachnoiditis of undetermined etiology." An autopsy was performed three hours after death.

Autopsy Findings.—No external lesions were found except for well healed, postoperative incisions in the posterior parietal region of the scalp. Examination of the abdominal and thoracic viscera revealed no lesions except for moderate pulmonary edema and evidence of acute inflammation in the dome of the urinary bladder. Both adrenals were sectioned and no lesions were found. The skull presented two postoperative burr holes in the posterior portion of the parietal bones measuring 5 mm. in diameter. Some clotted, extravasated blood in the process of resorption was present on the outer and inner surface of the dura in the immediate vicinity of the trephine openings. The brain weighed 1,450 grams. The hemispheres were symmetrical. The gyri were flattened and the sulci compressed. Two postoperative circular defects in the posterior portion of both parietal lobes plugged with pinkish-yellow, friable clots were seen. The leptomeninges covering the

FIG. 1

Case 1.—Photograph of brain. Note obliteration of anatomical landmarks around optic chiasm by thickened meninges and black mottling over left cerebellar hemisphere.

hemispheres were delicate and transparent. However, over the base of the brain and cerebellum, the meninges were diffusely thickened and opaque with some orange-grey, black and red-purple streaking *(Fig. 1)*. The thickening faded out laterally. The details of the circle of Willis and optic chiasm were obscured by the meningeal thickening. The appearance of the basilar lesion simulated that of tuberculous meningitis except for the coloring which appeared somewhat more vivid and variegated. The gasserian ganglia on both sides were edematous, soggy and showed grey and black discoloration. Their cut surface appeared gelatinous, greyish-black with creamy yellow, punctate spots scattered throughout. Step sections of the brain revealed marked dilatation of the entire ventricular system. The ventricles were filled with a large amount of colorless, clear, cerebrospinal fluid. No other lesions were noted. The pituitary, pineal and choroid plexuses appeared grossly normal. Only the upper cervical segment of the spinal cord was examined and showed diffuse thickening of the pia-arachnoid along the lateral columns.

Microscopic examination of the viscera confirmed the anatomic diagnoses of pulmonary edema and acute cystitis.

Sections of the cerebrum, cerebellum and spinal cord revealed invasion of the subarachnoid space by sheets of highly pleomorphic cells. No organoid structures could be made out but many of the cellular elements were arranged in rows and occasionally in small sheaves. The neoplastic tissue seemed to creep along the contours of the convolutions of the base of the brain and cerebellum *(Fig. 2)*, thinning out over the summits of the gyri and forming large pools in the sulci and in the recesses around the roots of the cranial, cervical and spinal nerves. Nowhere did it appear sufficiently bulky to elevate the arachnoid or distort the contours of the cerebral cortex. The stroma of the tumor was scanty but richly vascularized and no necrosis of the tumor tissue was noted. The individual cells had distinct borders and abundant cytoplasm. In many areas, single cells and large groups of cells were impregnated with brown pigment which failed to give a positive reaction for iron. The amount of pigment and size of the pigment granules varied within a wide range. Some of the cells were free of pigment while in others, the cellular details were obscured by coarse brown aggregates. The nuclei varied a great deal in size, shape and staining intensity. Some of them were round, oval, indented or crescent shaped. Many of them assumed grotesque shapes. A great many mono- and multinucleated giant cells were seen *(Fig. 3)*. Large eosinophilic nucleoli were seen in the cells where the chromatin structure was loose. In many others, the nucleolus was apparently obscured by compact masses of chromatin. Some of the sections showed tumor tissue surrounding blood vessels and invading the adventitia. Sections of gasserian gang-

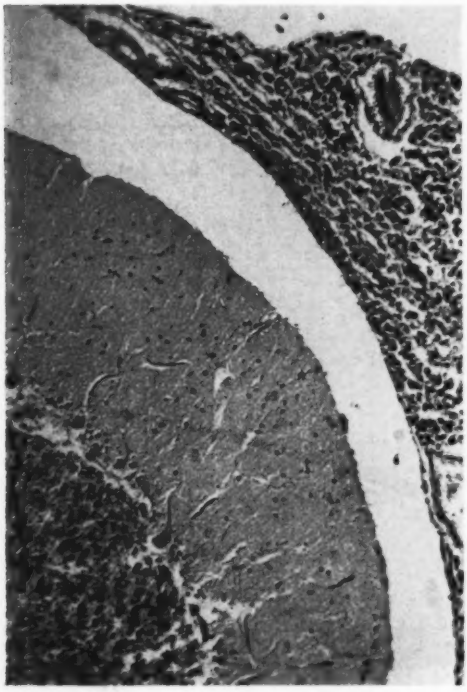

FIG. 2

Case 1.—Photomicrograph showing invasion of cerebellar leptomeninges by cellular neoplastic tissue (x 100).

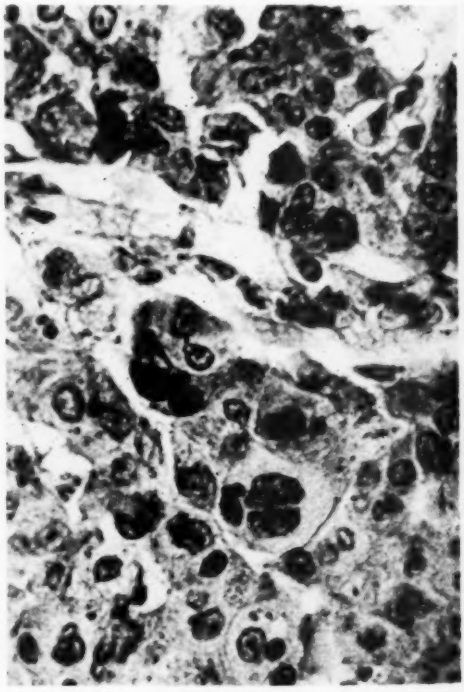

FIG. 3

Case 1.—Photomicrograph showing multinucleated, bizarre, tumor cells and aggregates of melanin in the cytoplasm (x 440).

lia showed diffuse invasion by tumor tissue *(Fig. 4)*. The tumor cells surrounded and displaced the nerve bundles emerging from the ganglia and were found in close apposition to the ganglion tissue itself. However, the boundaries between the tumor and nerve tissue remained clear cut and no invasion of the nerve fasciculi could be detected. The same distinct demarcation was noted between the brain cortex and the tumor in the subarachnoid space. Sections taken through the postoperative sites revealed disruption of the cerebral tissue, extravasation of blood with recent thrombi within the blood vessels and within the postoperative defect.

A section of the pituitary revealed a small nest of tumor cells within the capsule of the anterior lobe. It was separated from the pituitary tissue by a continuous narrow fibrous band.

CASE 2

History and Physical Findings.—A forty-one-year-old white miner was admitted to the Neurosurgical Section of the Veterans Administration Hospital, Richmond, Virginia in a critical condition. The family history was non-contributory. His relatives said that he had high blood pressure which was discovered in the course of a routine check-up about two years previously. However, he had no subjective complaints until about three months earlier, when he started complaining of "swimming in the head" upon severe exertion and some prefrontal headaches.

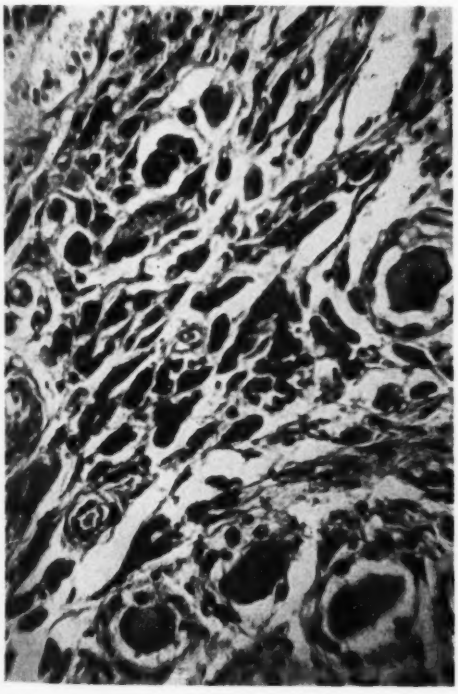

FIG. 4

Case 1.—Photomicrograph showing invasion of gasserian ganglion by heavily pigmented tumor cells (x 440).

About one month prior to his admission, he had an attack of syncope with loss of consciousness. It was observed that his head turned to the left and that he assumed a blank facial expression; about thirty minutes later, he collapsed and had a generalized tonic clonic convulsion. Two hours later he recovered from the seizure in a local hospital and was discharged after two days, apparently well except for generalized weakness and continuing headache. One week later, he began vomiting and was returned to that hospital where he showed marked fluctuation in his condition. He complained of pain in his legs and headache. He appeared restless and irritable. Three days prior to his admission, his speech became slurred and he developed paralysis of his arms and legs. His condition improved to a slight extent and he was transferred to our hospital.

Physical examination revealed a well developed, poorly nourished, slightly dehydrated white male, forty-one years old, who was restless, hyper-irritable, complaining of stiffness in his back, headache and pain in his legs. His speech was very slurred. He was not oriented as to time and place, but attempted to answer questions and to carry out requested maneuvers. The lungs were clear and resonant throughout, although the patient had a period of apnea and mild cyanosis. The heart was enlarged to percussion and auscultation. The apex beat was in the fifth interspace beyond the mid-clavicular line with a short systolic murmur. His blood pressure was 234/170 at the time of admission and fell to 190/134. The pulse was 128. The skin of the face was very red.

Both pupils were dilated. The left pupil was wider than the right. There was no reaction to light, accommodation or consensual stimulation. There was bilateral conjunctivitis, bilateral proptosis and bilateral ptosis, more marked on the left side. Extraocular movements were restricted in all spheres with bilateral paresis of 3, 4 and 6. Fundoscopic examination showed papilledema of 2 to 3 diopters with many hemorrhages and exudates in both discs as well as throughout the retina. The arteries and veins were not visible. There was marked streaking of the disc edges. Corneal reflexes were normal and there was normal sensation throughout the face. There was bilateral, central facial weakness, more marked on the right side. Hearing appeared to be normal, although on the right somewhat weaker than on the left. Swallowing was impaired. The tongue was deviated to the right without tremor. There was bilateral weakness of all extremities, more pronounced in the left arm. All superficial and deep tendon reflexes were absent except for the right biceps. Babinski was positive on the right and left. There was marked nuchal rigidity and positive Kernig and Brudzinski signs. The clinical impression was that the patient suffered from malignant hypertension with hypertensive retinopathy and hemorrhages into the pons, possibly from a ruptured aneurysm of the basilar artery with subarachnoid hemorrhage.

Laboratory Findings.—The urine showed a 3 plus albumen with many hyaline and granular casts, many epithelial cells and white blood cells in the sediment. The blood count and hemoglobin were within normal range. The sedimentation rate was 32; blood urea nitrogen was 14; serum protein was 5.6 grams

per 100 cc. with 3.5 grams per 100 cc. of albumen and 2.1 grams of globulin. Serologic tests for syphilis were negative. The spinal fluid pressure was 600. The fluid was grossly bloody and the serologic tests for syphilis gave a negative result. The white blood cell count 5 days after admission was 6,800 with 83 per cent neutrophils, 16 per cent lymphocytes and 1 per cent eosinophils.

X-ray examination of the skull revealed no evidence of increased intracranial pressure. The sella turcica appeared normal and the pineal could not be identified. The chest showed mottled densities in the supradiaphragmatic portions of both lung fields. The roentgen impression was that of basilar congestion or aspiration. The cardiac shadow was at the upper limits of normal.

Radiologic examination performed 8 days after admission showed numerous circumscribed areas of increased density scattered throughout both lungs which suggested metastatic neoplasm in both lungs.

Course in Hospital.—For the first week, the patient showed a remarkable improvement. His speech became intelligible. He became mentally alert and oriented as to time, place and person. His left facial paresis was much improved to the point where he could close his left eye, but the right facial paresis remained unchanged. The extraocular movements became almost normal. The left pupil remained larger than the right, but both reacted slightly to light, accommodation and consensual stimulation, although the papilledema remained unchanged. There was also marked improvement in his shoulder, arm and leg movements, although they remained weaker on the left. He could not swallow for the first few days, but cooperated in tube feedings. Six days after admission he removed his gastric tube and was able to swallow his meals. His left Babinski remained but his right disappeared. The abdominal reflexes returned but not the deep tendon reflexes. His blood pressure was unstable but tended to become lower and to stabilize around 160/120. On the eighth hospital day the patient became restless and confused during the night. A lumbar puncture revealed a pressure of 300 mm. with a dark amber fluid containing some fresh blood. A recurrence of the intracranial hemorrhage was suspected. His temperature during the first week in the hospital fluctuated between 100 and 102°F., but came down to around 99°F. after administration of penicillin. Three successive lumbar punctures revealed a spinal fluid pressure ranging from 470 to 240 mm. The spinal fluid contained a slight amount of blood and the supernatant fluid became progressively lighter yellow. His blood pressure started to rise slowly beginning with his fifteenth hospital day and reached 190/140 on the seventeenth hospital day. A lumbar puncture performed on the same date showed a grossly bloody spinal fluid. The patient became comatose and died on the eighteenth hospital day. An autopsy was performed four hours after death.

Autopsy Findings.—Examination of the brain revealed some flattening of the cerebral gyri. The meninges had a dark yellowish-brown cast. The right parietal lobe was bulging and fluctuant. A

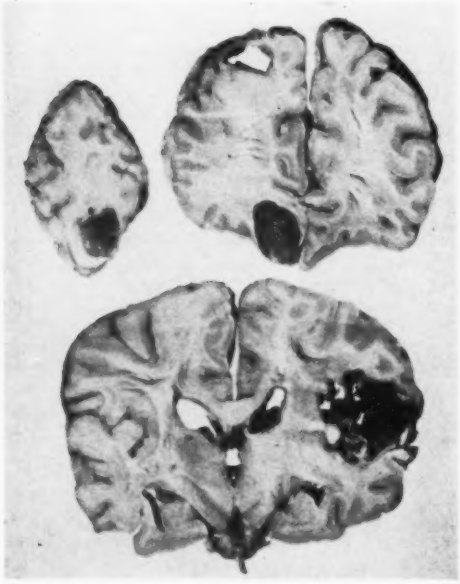

FIG. 5

Case 2.—Coronal sections of the brain showing primary site of the tumor in the right parietal lobe and smaller metastatic implants in left frontal and left occipital lobes.

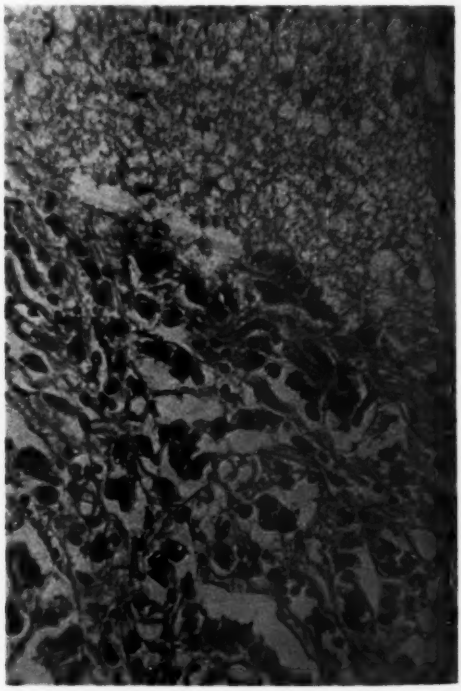

FIG. 6

Case 2.—Photomicrograph showing invasion of cerebral tissue in the periphery of the primary tumor (x 200).

rounded, dark brownish-black, friable mass measuring 4 x 4 x 3 cms. occupied the region of the right insula extending medially from the meningeal surface *(Fig. 5)*. It distorted the insula and the lateral contour of the right hemisphere. A dark brown, oval shaped nodule of a somewhat spongy consistency was found in the left frontal lobe near its lower surface. Several smaller, pigmented, round and oval nodules measuring from 5 to 10 mm. were scattered through the brain substance. All of the nodules came in contact with the leptomeninges and revealed various intensities of pigmentation. Their consistency was somewhat firmer than that of the brain.

In addition to the cerebral lesions, several visceral implants were found. A discrete nodule 4 mm. in diameter was found in the myocardium of the right ventricle. The lungs were studded with numerous nodules varying in diameter from 0.5 to 1.2 cm. Seven similar nodules were found in the lining of the jejunum and two in the cortex of the right kidney. In view of these findings, the entire body surface was searched for any lesions which could be interpreted as a primary site of a melanoma. The search included the scalp, nail beds, interdigital spaces and perineum. A small flat elevation on the back between the shoulder blades was sectioned and revealed a skin polyp. The posterior half of both eyeballs was removed and the contents of both eyes were examined grossly and microscopically with negative results.

No other significant pathologic findings were noted throughout the body with the exception of some trivial atheromatous plaques in the aorta. Both adrenals were examined grossly and microscopically for melanomatous foci.

Microscopic examination of the tumor nodules revealed some variation in the morphologic pattern of the tissue as well as of the individual cells in sections taken from various sites. All of them were within range of morphologic variants which any malignant tumor, and particularly a melanoma, might display when implanted in different host tissues. The largest tumor mass appeared to invade the brain tissue adjacent to the meninges *(Fig. 6)*. There was also a great deal of hemorrhagic extravasation in its periphery with some extravasated blood present in the subarachnoid space beyond the periphery of the tumor. The other meningeal lesions seemed to displace rather than invade the cerebral tissue *(Fig. 7)*. They were made up of sheets of fairly uniform, rounded or polygonal cells with distinct cell borders, moderately rich cytoplasm and vesicular nuclei of varying density. The nucleoli were prominent in some of the cells and missing in most of the nuclei. Mitotic figures were numerous and some of them were atypical. The degree of impregnation with

FIG. 7

Case 2.—Photomicrograph showing displacement of cerebral tissue by the tumor implants. Note zone of impregnation by pigment (A) (x 60).

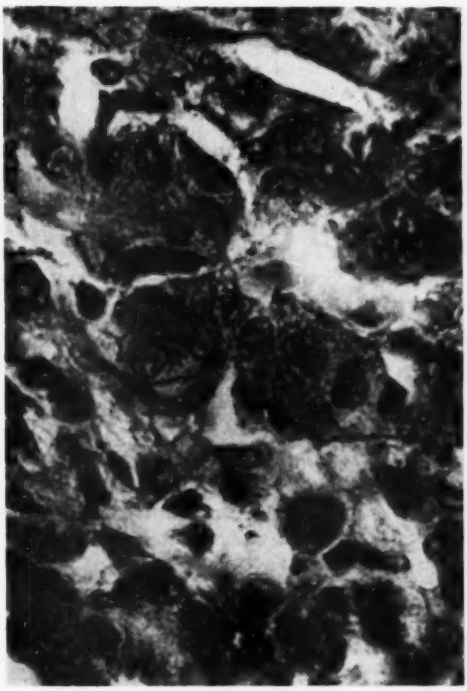

FIG. 8

Case 2.—Photomicrograph of a pulmonary implant showing pleomorphism of the tumor cells and pigment aggregates (x 440).

melanin pigment varied in different areas, from very heavy to barely perceptible, and some fields appeared entirely free of pigment. Numerous microcystic spaces, probably due to degeneration, appeared within the tumor. The cells in the immediate vicinity of the cysts were arranged in concentric rows but this arrangement faded imperceptibly into solid sheets.

The tumor nodules in the lung, heart, kidney and intestine displayed a higher degree of pleomorphism *(Fig. 8)*. The cells were larger with more abundant cytoplasm and assumed polygonal shapes. The pigment was found in fine and coarse granules which, in many cells, formed compact aggregates obscuring the structural details of the cells. In addition to the pigment in the tumor cells, a great deal of pigment was found within macrophages. The stroma of the tumor was scanty and well supplied with blood channels. The host tissues showed evidence of displacement and compression by the tumor nodules.

The first of the two cases presented by us meets the criteria for diagnosis of a primary melanoma of the meninges inasmuch as no other foci of neoplastic tissue were found elsewhere in the body and the tumor formed a diffuse melanoblastic infiltration.

The rapid course of the illness and the histologic pattern of the neoplastic tissue leave no doubt as to the malignant character of the disease. Although the orbital contents were not examined in the course of the autopsy, numerous examinations of the eye grounds performed while the patient was hospitalized rule out the possibility of a primary ocular melanoma. Melanin bearing cells are normally found in the meninges, over the spinal cord and in the recesses and sulci of the brain and cerebellum. This makes it a likely area for formation of primary melanotic new growths. The clinical course was strongly suggestive of a meningitic process. The only findings which were inconsistent with the diagnosis of meningitis of inflammatory nature were the paucity of cells in the cerebrospinal fluid in the presence of a high protein content. This dissociation of cells and protein and the consistently negative bacteriologic findings were the only clinical features which should have cast some doubt on the diagnosis of meningitis due to bacterial agents. The air studies which were performed because a tumor was suspected in view of the increased spinal fluid pressure, papilledema and bradycardia, were non-contributory and the diagnosis of tumor therefore was abandoned.

The second case formed a different anatomic and histologic pattern and, as a result of this, the clinical course suggested the diagnosis of intracranial focal lesions. The clinical impression was that of multiple cerebral hemorrhages. Hypertension, retinal changes, original chest x-ray film, as well as the laboratory findings, pointed to the diagnosis of vascular disease of the brain. The second x-ray film of the lungs 10 days prior to death could have suggested a neoplastic process, although melanoma could not be suspected.

We assume that the primary growth was located in the lateral portion of the right cerebral hemisphere *(Fig. 4)*. Our assumption is based on the following observations.

(1) The lesion was conspicuously larger than all other implants in the cerebrum.

(2) It showed areas of necrosis and microcystic degenerative changes which seem to place the origin of the lesion at an earlier date than all the other nodules which were made up wholly of viable neoplastic cells.

(3) The metastatic foci in the lungs appeared on the radiologic examination when the illness was pretty far advanced. The lesions in the heart and jejunum were still smaller than the lesions in the lung and we believe they were formed after the implants of the pulmonary vascular tree took place.

(4) None of the foci occurring extracranially were found in melanogenic regions. The heart and kidney are also considered as unlikely sites for primary melanotic tumors.

A discussion of the pathogenic theories and classification of melanotic new growths of the meninges is beyond the scope of this paper.

SUMMARY

The clinical and pathologic findings in two cases of primary pigmented meningeal melanoma are reported. One of the cases represents a diffuse form simulating chronic meningitis without metastatic spread. The other case is a nodular form with metastases to other viscera. The difficulties in arriving at a clinical diagnosis are discussed and the literature is reviewed.

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DISCUSSION *(Abstract)*

Dr. Bela Halpert, Houston, Tex.—I have treated two cases of malignant melanoma that apparently arose in the glomus chorioideum of the lateral ventricle. One of these was in a 25-year-old woman. The primary growth was in the left glomus and produced a single metastatic nodule 7 cm. in diameter in the left lung. This case was described by Wilkins and me in the Journal of the Oklahoma State Medical Association in 1946 (3:9 pp. 503-506). The other growth was in a 55-year-old man, a patient in the Veterans Administration Hospital, Houston, Texas. In this patient the growth also arose from the left glomus chorioideum and caused extensive metastases in the brain alone and none elsewhere.

Melanoblasts are distributed everywhere where ectoderm touches mesoderm. Since the central nervous system is developed of ectoderm and melanoblasts are present in the leptomeninx it is not unexpected that malignant melanomas should arise in the leptomeninx.

Dr. Charles G. Bratenahl, Key West, Fla.— I wonder if Dr. Russi would explain whether there might be any relationship to the brown pigmentation one often sees around the meninges at the base of the brain and its chemical nature.

Dr. Russi (closing).—Thank you very much, Dr. Halpert, for bringing up the point about the presence of melanophores in meninges.

Melanophores are present all through the meninges, particularly in the deeper portions of the sulci and recesses of the leptomeninges. Condensation of these melanin-laden cells is responsible for the brown pigmentation around the base of the brain referred to by Dr. Bratenahl.

According to some authors, melanophores are more numerous around the portions of the central nervous system where the circulation of the cerebrospinal fluid is sluggish and some tendency to stagnation exists. Such is the situation around the base of the brain and in the deeper portions of the sulci.

Melanophores are apparently more numerous during childhood. They seem to be less frequent in adult bodies, although their presence is quite common even then.

As Dr. Halpert pointed out, melanomas are to be expected wherever ectoderm and mesoderm come in contact and there is no reason why they should not arise more frequently from the meninges. The literature on the subject, however, is quite scanty. There are not over 65 cases reported including even some sketchy reports in the foreign literature which were not available to us at the time of preparation of the manuscript.

VENOUS CATHETERIZATION OF THE HEART IN SELECTION OF PATIENTS FOR MITRAL COMMISSUROTOMY*

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With the advent of successful mitral surgery[1-7] renewed interest in the pathological physiology of mitral stenosis has been stimulated. Thirty-six patients with predominantly mitral stenosis have had cardiac catheterization studies performed, and thirty of these patients have been operated upon to date with two deaths. It is the purpose of this paper to present the findings of the catheterization and the possible indications for operation as determined by this method.

Methods.—Venous catheterization of the right side of the heart was performed in the usual manner under fluoroscopic control. Arterial oxygen samples, using heparinized, mercury sealed syringes, were obtained from the brachial, femoral or radial arteries and the mixed venous samples were drawn from the

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pulmonary artery or right ventricle. Oxygen content was determined at rest and with exercise by the Scholander[8] micro-technic methods or the manometric method of Van Slyke.[9] Pressure readings were recorded from the so-called "pulmonary capillary" area, pulmonary artery, right ventricle, right atrium and arterial sites by means of a Sanborn electromanometer and a direct writing polyvisocardiette. Determinations were made during rest and during exercise, the latter consisting of six to eight minutes of bicycling type of movements with the lower extremities at a rate of 20 to 25 alternate pumps per minute. Cardiac output was determined by the Fick principle[10] with the patient at rest and during exercise, and in many of the patients ten minutes after the completion of the exercise.

RESULTS

The results of the venous catheterization of the heart may be summarized in the following table:

DISCUSSION

Abnormal function in mitral stenosis results from a mechanical obstruction to the blood flowing from the left atrium to the left ventricle from residual pulmonary interstitial fibrosis which accompanies rheumatic fever and from rheumatic myocardial involvement. These factors tend to increase pulmonary vascular pressures and this is confirmed by cardiac catheterization.

Resting pulmonary arterial pressures were found to be routinely elevated above the normal mean pressure of 15 mm. of mercury and increased markedly during exercise. In two patients during exercise the mean pulmonary arterial pressures were found to be above 90 mm. of mercury, each having a very incapacitating type of disease. In a high percentage of the patients, the pulmonary arterial pressure almost doubled with exercise unlike normal individuals in whom there is little or no increase with exercise. Excessively high pulmonary arterial pressures were found in the more advanced states and in older patients. Baker *et alli*[5] have emphasized that a low resting pulmonary arterial pressure with a sharp rise after exercise is encountered in

CARDIAC CATHETER FINDINGS IN 36 CASES OF MITRAL VALVULAR HEART DISEASE

Clinical Classification	Normal Values	II (2 pt.)		III (18 pt.)		IV (15 pt.)	
		Range	Average	Range	Average	Range	Average
"Pulmonary capillary" mean pressure (resting) mm. Hg.	8-10	9-12	10.5	13-30	23	19-56	28.5
"Pulmonary capillary" mean pressure (exercise) mm. Hg.	8-10	15-16	15.5	13-52	30	23-46	35
Pulmonary artery mean pressure (resting) mm. Hg.	10-15	14.5-18	16.2	12-44	30.2	26-70	51
Pulmonary artery mean pressure (exercise) mm. Hg.	10-15	21-24	22.5	34-90	46	36-103	63
Arteriovenous oxygen difference (resting) volume per cent	3.5-6.0	3.9-4.4	4.15	1.9-6.8	4.7	3-10.2	5.9
Arteriovenous oxygen difference (exercise) volume per cent		5.7-7.6	6.7	1.9-12.5	7.2	5.1-13.0	9.6
Cardiac output (Fick principle) (resting) liters/minute		5.1-6.7	5.9	2.8-6.4	4.2	1.7-5.8	2.9
Cardiac output (Fick principle) (exercise) liters/minute		5.8-6.6	6.2	3.0-8.2	6.0	1.4-6.7	3.8
Cardiac index (resting) liters/minute/M^2	3.31+ or —0.5	3.1-3.8	3.4	1.6-4.7	2.5	1.6-4.0	2.5
Cardiac index (exercise) liters/minute/M^2		3.5-3.8	3.65	1.7-5.6	3.1	0.7-3.6	2.4
Arterial oxygen (resting) per cent saturation	95	81-94	87.5	63-90	81.0	60-88	79
Mitral valve size (Gorlin & Gorlin calculation) $cm.^2$	4.0-6.0	1.74-2.94	2.34	0.9-1.8	1.17	0.48-1.41	0.95
Pulmonary arteriolar resistance (resting) dynes/second/$cm.^5$	80-100	46-107	76	33.497	181	37-914	265
Pulmonary arteriolar resistance (exercise) dynes/second/$cm.^5$		68-127	97	66-535	220	66-1028	295
Total pulmonary resistance (resting) dynes/second/$cm.^5$	250-300	214-224	219	267-1071	621	384-1828	798
Total pulmonary resistance (exercise) dynes/second/$cm.^5$		290-291	290.5	268-1264	609	755-4129	1774

TABLE 1

individuals before pulmonary hypertension is far advanced.

In the resting state, we were able to catheterize the so-called "pulmonary capillary" area in thirty-one patients. In twenty-one, the mean pressures were between 20 and 30 mm. of mercury; four were over 30; and three were over 40 mm. of mercury. All of the latter group were in pulmonary edema at the time of catheterization. Three of our series had normal pressures of less than 8-10 mm. of mercury, the mean pressure. During exercise, five patients had pulmonary capillary pressures between 30-35 mm. of mercury, and two went into pulmonary edema with onset of coughing, orthopnea and moist rales. Eight of twenty patients had mean pressures above 40 mm. of mercury and went into pulmonary edema. Acute pulmonary edema has been found to develop at a range of 35 mm. of mercury mean pressure by other workers.[11 12] Ellis *et alii*[11] have noted that it is more prone to occur in a patient with sinus rhythm and a cardiac index of less than 3 liters per minute, and our observations have borne this out. It is known that the pulmonary veins have no valves and, therefore, with left atrial failure, a greater back pressure occurs in the pulmonary vascular bed in as much as there is no buffering mechanism between the mitral stenotic valve and the pulmonary capillary. The pressure in the pulmonary veins, left atrium and pulmonary capillary are quite similar.[13] Accordingly, the increased pressure in the left atrium is immediately reflected in the pulmonary circuit and when a rise to the critical level of about 35 mm. of mercury occurs, then pulmonary edema may result. Tachycardia occurring on exertion may increase the blood flow into the pulmonary circuit and also precipitate pulmonary edema.[12] We found, as other workers have,[11 12] that when the pulmonary capillary pressure reached the mean of 30 to 35 mm. of mercury, corresponding increase in pulmonary arterial pressure was more marked. It is thought that this is a protective mechanism brought about by the increased pulmonary arteriolar resistance in order to keep the pulmonary capillary pressure from increasing markedly. In contrast to patients with mitral stenosis, normal subjects have little or no increase in either the pulmonary capillary or pulmonary arterial pressures with exercise. Pressure tracings obtained in the "pulmonary capillary" area were of further value because in some a prominent or spiking "v" wave was noted *(Fig. 1)* demonstrating the presence of an associated mitral insufficiency. Generally flat curves and small "v" waves in the pulmonary capillary bed indicate that little or no mitral regurgitation is present. Gorlin *et alii*[12] attempted to correlate the height of the "v" wave with the degree of mitral insufficiency, but our data were insufficient for this correlation to be adequately demonstrated. Right ventricular pressures were found to show elevation of the systolic pressure. Diastolic right ventricular pressures were noted to be zero unless right ventricular failure was present, in which case there was elevation of diastolic pressure. Right atrial pressures were measured and the majority were below a mean of 5 mm. of mercury. In one individual in whom there was right ventricular failure at the time of cardiac catheterization, right atrial pressures of 17.5/12.5 mm. of mercury were recorded.

Arteriovenous oxygen difference which represents the peripheral extraction and utilization of the oxygen is inversely proportional to the blood flow. The normal resting arteriovenous difference has been found to be 4-5 volumes per cent and is never above 6.0 vol-

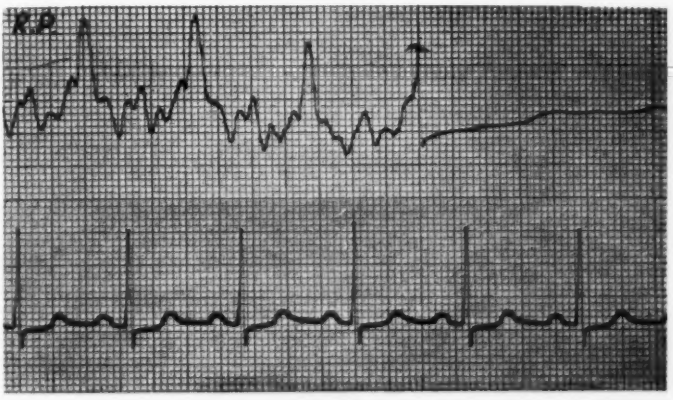

FIG. 1

Prominent V waves seen on "pulmonary capillary" pressure tracing. The pulmonary capillary mean pressure was 13 mm. of mercury.

umes per cent.[11] In one-third of our patients the resting arteriovenous oxygen difference was found to be above 6 volumes per cent, and in eight, following exercise, an increase to over 10 volumes per cent was noted suggesting a very marked extraction of oxygen in peripheral circulation together with a decreased cardiac output.[11] In fifteen patients in whom the arterial oxygen saturations were below 90 per cent, there was marked exertional dyspnea. A patient with 63 per cent saturation was totally bedfast, two were in the range of 78-80 per cent, and the others were between 80-88 per cent. Andrus[14] has stated that the diminished resting arterial oxygen saturation suggests that changes in the alveolar walls secondary to chronic capillary congestion interfere with diffusion and these changes may not be reversible with valvulotomy. Other factors producing chronic dyspnea may include pulmonary parenchymal disease, interstitial fibrosis and pulmonary arteriolar sclerosis.

Initial oxygen consumption was somewhat reduced in these patients with mitral stenosis but oxygen consumption doubled on exercise in the majority of these patients.

Cardiac outputs were variable depending upon the functional capacity of the individual. Using a level of 3.31+ or —0.5 liters per sq. meter per minute as normal values,[11] we found that in patients with functional class 4 disability, there was a marked reduction in resting output with little or no increase with exercise. In four cases a further lowering with exercise occurred. Three of these went into pulmonary edema during exercise. However, in several others pulmonary edema was precipitated with exercise, but there was some increase in cardiac output. In class 2 and some individuals in class 3, normal outputs with good increases with exercise were found. However, the cardiac outputs were quite variable in these classes. In general those patients who were manifesting auricular fibrillation had lower cardiac outputs than those individuals with normal sinus rhythm. This is thought to be due to the fact that in severe mitral stenosis, a large percentage of ventricular filling may be accomplished by auricular systole as observed by Ellis.[11] With tachycardia in mitral stenosis there is a decrease in diastolic filling time with a subsequent decrease in cardiac output, and the increased rate cannot make up for the decreased ventricular filling. The decreased size of the mitral orifice and the increased peripheral extraction of oxygen by the tissues further decreases cardiac output.

The normal mitral valve has a cross sectional area of 4-6 sq. cm. as calculated by the formula of Gorlin and Gorlin.[15] There is considerable controversy as to the direct applicability of the mitral valve size in relation to prognosis and selection of patients for surgery, but we have attempted to calculate it in our patients and have found relatively good correlation with operative and autopsy studies. Dexter[13] believes that surgery may not be justified if the valve area is over 1.5 cm. sq. Fairly good correlation between the decreased mitral valve size and the decreased cardiac output was noted. "Pulmonary capillary" pressures and pulmonary arterial pressures were found to be markedly elevated in those patients with narrowed mitral orifices.

Pulmonary arteriolar resistance as calculated by the formula:[12] pulmonary arterial resistance minus "pulmonary capillary" pressure divided by cardiac output times 1332 dyne seconds cm.^{5} normally is 70-80 dynes seconds cm.^{5} In over half of the patients in which this determination was made, there was rather marked increase in it. But the correlation with mitral valve size or functional capacity was only fair. Rheumatic interstitial fibrosis which may alter the distensibility of the lung and in turn the vascular system as well as the pulmonary arteriolar intimal thickening may account for this increased resistance. Increased total pulmonary resistance as calculated from the formula:[12]

$$\frac{\text{Pulmonary artery}}{\text{Cardiac output}} \times 1332 \text{ dyne seconds cm.}^5$$

in general follows the increase of pulmonary arteriolar resistance in our patients. Parker[16] says that in his series, those patients with no increase in pulmonary arteriolar resistance frequently revealed exertional dyspnea, orthopnea, hemoptysis and acute pulmonary edema with no venous distension and no right ventricular failure present. In those patients with mild increases in pulmonary resistance, a delicate balance in which the heart could not pump the blood in any faster than it could be handled resulted in a low fixed cardiac output and the symptoms of fatigue, exhausted feeling and weakness and tiredness

prevailed. In the individuals with an excessively high pulmonary resistance, excessive exertional dyspnea, orthopnea and right ventricular hypertrophy failure were seen.

Indications for valvulotomy as ascertained from venous catheterization of the heart, which, of course, must be used in connection with a careful clinical evaluation of the patient, may be summarized as:

(1) Elevated resting "pulmonary capillary" pressure increasing with exercise.
(2) Elevated resting and exercise pulmonary arterial pressure.
(3) Reduced resting cardiac output with little or only slight increase during exercise.
(4) Arteriovenous oxygen difference elevated above normal, both in the resting and exercise states.
(5) Increased pulmonary arteriolar resistance and total pulmonary resistance.
(6) Decreased mitral valve size as calculated by the formula of Gorlin and Gorlin[15] (below 1.5 cm.^{2}).

As our experience in clinical correlation with cardiac catheterization grows, we believe that clinical evaluation may supplant venous catheterization in evaluation of patients for mitral commissurotomy.

SUMMARY

Thirty-six cases of rheumatic heart disease have been studied by means of venous catheterization of the heart and cardiac output determination. Thirty have had commissurotomies with two deaths. Indications for commissurotomy as suggested by cardiac catheterization have been presented.

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STEROID HORMONES AND THE COLLAGEN DISEASES*

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Four years have elapsed since Hench[1] administered compound E to a patient with rheumatoid arthritis. Since that time patients with many and varied diseases have been given cortisone and adreno-corticotrophic hormone. The results of this therapy have been dramatic and at the same time have been disappointing. Patients suffering from diseases that heretofore were crippling or progressively fatal have been given a reprieve, although in most instances this reprieve is for short periods of time. The immediate beneficial results from the use of these substances are truly miraculous but the long-term effects are difficult, in fact, impossible to evaluate.

During the four years that ACTH and cortisone have been available, they have been given to patients with many diseases and perhaps their greatest usefulness has been in those people who have collagen diseases. This discussion is limited to those diseases that are generally considered to fall in the group of collagen diseases.

Klemperer[2] has pointed out that,

"The term diffuse collagen diseases was originally applied to acute and chronic maladies which are

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characterized anatomically by generalized alterations of the connective tissue, particularly by abnormalities of its extra-cellular components. In this sense the term can include rheumatic fever, rheumatoid arthritis, polyarteritis, acute lupus erythematosus, generalized scleroderma, and dermatomyositis."

Others have included clinical entities in addition to those enumerated by Klemperer. Because of the similar connective tissue changes, Klinge[3] has included malignant nephrosclerosis, thromboangitis obliterans, subacute bacterial endocarditis and certain nephritides. Similar connective tissue changes have been produced experimentally by employing varying technics. In some instances hypersensitivity reactions have been employed although the lesions have been noted when the possibility of hypersensitivity was excluded. It is important to point out that the alterations in the connective tissues are observed in many conditions which have not a common pathogenic factor. Furthermore in tissue preparations for study of microscopic changes many instances of overlapping of the characteristic pathologic picture have been noted.

For instance, the vascular lesions of acute lupus erythematosus may simulate those of periarteritis nodosa. Clinically these diseases may have a rather characteristic pattern so that the diagnosis is usually made with a fair degree of accuracy. Recently, however, attention has been called to the entity of thrombotic thrombocytopenic purpura,[4] which may simulate to an alarming degree disseminated lupus erythematosus, both from a clinical and pathological standpoint. The demonstration of L. E. cells by Hargraves *et alii*,[5] aids greatly in the diagnosis of lupus erythematosus.

For the purposes of this discussion we have arbitrarily accepted those diseases originally enumerated by Klemperer[2] as being collagen diseases.

Until January 1, 1952, 315 patients had been treated at Duke Hospital with ACTH or cortisone. Of these, 114 had one of the collagen diseases. The diagnosis was based upon the characteristic clinical and laboratory studies. When indicated biopsy studies were performed.

Rheumatic Fever.—Because of the relative infrequency of rheumatic fever in North Carolina as compared with other communities we have had little opportunity to follow these patients. To evaluate the efficacy of therapy in chronic rheumatic fever is beyond the scope of a general hospital. Young and Rodstein[6] have reported a series consisting of eight patients and they conclude that certain beneficial effects were attained which could be attributed directly to ACTH or cortisone and in some of the children with severe disease they believe that the prolonged administration of large doses was probably life saving. We have had experience with only two patients with chronic rheumatic fever.

In the acute phase we have treated 15 patients and are able to confirm the observations of others.[7] There is usually an immediate improvement subjectively and objectively, with subsidence of fever and acute joint symptoms. Remissions were obtained in all but one patient treated although the tendency to recurrence was ever present. We believe that during the acute phase of severe rheumatic fever these drugs are of considerable value.

RHEUMATOID ARTHRITIS

Critical reviews[1] have appeared recently on the therapy of rheumatoid arthritis and in view of the experience at Duke Hospital we believe that we are able to confirm the observations of others. All are familiar with the prompt response of the constitutional symptoms of fever, anorexia, weakness, and pain, swelling and stiffness of the joints. The improvement in certain aspects of the pathologic picture of rheumatoid arthritis following the use of ACTH and cortisone has been demonstrated by Norcross and associates.[8] These workers were not able to correlate the severity of nodular polymyositis or subcutaneous nodules with activity or therapeutic response of the rheumatic process. These observations are of great interest and show promise of providing further understanding of the underlying disease process. The flexion contractures in the more chronic cases may be greatly relieved although no improvement has been noted in the deformities of cartilage or bone or when the contractures have become fibrosed. So long as these drugs are administered the remission is maintained; however, cessation of administration is followed by a relapse of the symptoms. The

remission may continue for only a few days to several months after the drugs have been discontinued.

Emphasis must be laid upon the observation at Duke Hospital and by other investigators, that all patients with rheumatoid arthritis do not obtain a remission following the administration of ACTH or cortisone. Two patients have been seen in this clinic who presented some of the physiological effects of over treatment, who had no subjective or objective improvement in the arthritis.

ACTH and cortisone have been used in combination with other drugs, notably para-aminobenzoic acid[9] and gold.[10] This form of therapy may hold certain promises that have not yet been realized with ACTH or cortisone alone.

Dermatomyositis.—This rare disease has been seen and treated in two patients at Duke Hospital. No beneficial effects were noted in either patient although the dosage administered was thought to be adequate. This experience has been similar to that of others, although, Kierland *et alii*,[11] reported a patient in whom a marked degree of improvement was noted and in whom the remission continued on a daily dose of 50 mg. of cortisone.

Scleroderma.—Nine patients with scleroderma have been treated with ACTH or cortisone at Duke Hospital with no beneficial results. Kierland and associates[11] reported that in patients whose disease was of short duration there was perhaps more flexibility of the fingers, wrists and jaws. Rosenberg and associates[12] report no improvement in two cases. In one case treated here, the patient thought that the movements of his hands were improved but objectively no improvement was noted.

Polyarteritis.—ACTH and cortisone in what was considered adequate dosage were given to four patients with polyarteritis. The diagnosis in each instance was considered accurate as shown by biopsy. Except for some euphoria and a sense of well being during the period of administration, no beneficial results were observed. This observation is more or less in line with that of other investigators, although improvement has been noted by some. In the four patients no history of drug sensitivity could be elicited.

Disseminated Lupus Erythematosus.—In the treatment of the patient with disseminated lupus erythematosus, corticotropin or cortisone has in some instances been dramatic and life saving. Twenty-seven patients have been treated at Duke Hospital and in two of these no remission was obtained in spite of adequate dosage. On the other hand, four of these patients had remissions that lasted six months or longer without continuing therapy. This, we believe, is significant although it is a well recognized fact that remissions occur without treatment. DuBois *et alii*[13] have shown that spontaneous remission occurs in the untreated patients in 20 per cent of cases, whereas in the treated patients remissions occur in nearly all cases depending upon the dosage of corticotropin or cortisone. In our group in the two patients who failed to respond, that is, failed to have a remission, the disease was far advanced and the patients were practically moribund when treatment was instituted.

Eight of the patients in this group have died; six of these were from overwhelming infections. While the life expectancy of patients with disseminated lupus erythematosus is not increased either in the group observed at Duke Hospital or in other reported series[13 14] it is shown that remissions are produced and the patients are improved temporarily. It is of interest that one patient had acute appendicitis with a successful appendectomy. Another patient conceived and delivered a normal infant during a remission induced by corticotropin and cortisone therapy.

Complications of Therapy.—Complications arising from the use of corticotropin and cortisone are always present and with few exceptions are manifestations of the physiological effects. These complications for the most part are dependent upon the dosage or the amount of the substances administered. Sprague[15] has written an excellent critical review emphasizing the physiological effects of cortisone and ACTH and a clearer understanding of the complications can be obtained from the study of this paper. Other factors than the dosage, of necessity, enter

into determining the incidence and severity of the complications. The age of the patient bears a most important relationship since in children serious reactions may appear without premonitory sign. The illness with which the patient is afflicted will determine to a great extent, the nature of the untoward effect. For instance, in acute disseminated lupus erythematosus, renal involvement, convulsions and psychoses may intervene, before adequate control of the disease has been attained by ACTH or cortisone. Russell, Haserick and Zucker[16] have emphasized the frequency with which epilepsy occurs in lupus erythematosus. We believe that in most patients, as of this date, the majority of complications can be avoided by carefully following the patients, and whenever a reasonable doubt exists as to the exact state of the patient the drugs should be discontinued. Certain precautions must be observed, such as requiring a low sodium intake and the administration of potassium salts in adequate amounts.

The signs and symptoms of Cushing syndrome are perhaps the most frequently observed in patients receiving corticotropin and cortisone. The appearance of edema, acne, hirsutism, fullness of the face, hypertension and glycosuria are observed not infrequently and are not in themselves contraindications for continuation of therapy. Nor for that matter do these symptoms necessarily call for a reduction in the dosage if the primary disease warrants further treatment. Great caution must be exercised however, that these symptoms do not become too severe.

Hypochloremic hypokaliemic alkalosis must be anticipated and prevented when therapy calls for large dosage and continued administration. Frequent estimations of the concentration of these electrolytes in the serum must be carried out and at the first indication of a decrease in the concentration adequate replacement must be instituted and administration of corticotropin and cortisone stopped.

As a precautionary measure 10 to 30 ml. per day of a mixture consisting of one gram of each of the following: potassium acetate, potassium bicarbonate and potassium citrate per 8 ml. of water is of benefit in the prevention of this dangerous electrolyte imbalance. Potassium chloride may also be given.

Gastro-intestinal disorders have received less attention than is warranted. Perhaps one of the most frequent symptoms complained of by the patient receiving cortisone is epigastric pain. This pain may become so severe that further administration of the drug is prohibited although in most instances a reduction in the dosage will stop the pain. Gray and associates[17] have shown a tremendous increase in the gastric secretion in patients receiving corticotropin or cortisone in moderate dosage for several weeks. It is believed that this is responsible for pain from which these patients complain. Severe gastric hemorrhage[18] and intestinal perforations have been reported. We have not observed these conditions in our patients.

Pathologic fractures [19] have been reported in patients with rheumatoid arthritis who have been treated with cortisone for long periods of time. While it could not be concluded definitely that the osteoporosis resulting from the administration of cortisone is responsible for the fractures, it is highly suggestive.

Dosage and Administration.—Little can be said regarding the dosage of either corticotropin or cortisone since there is a variation in the amounts necessary to produce and maintain a remission from the disease for which these substances are administered. In recent months ACTH has been administered by continuous intravenous drip over a period of from eight to twenty-four hours. The reduction in dosage is achieved by reducing the time that ACTH is allowed to flow. Twenty units of ACTH are dissolved in one liter of 5 per cent dextrose solution. Cortisone is administered by mouth whenever possible

RESULT OF TREATMENT WITH ACTH AND CORTISONE

	Number of Patients Treated	Improvement	Minimum Improvement	No Improvement	Six Months Remission
Rheumatic fever	21	20		1	
Rheumatoid arthritis	51	47	2	2	3
Acute lupus erythematosus	27	25		2	4
Polyarteritis	4	0		4	
Dermatomyositis	2	0		2	
Scleroderma	9	0		9	

TABLE 1

and in amounts necessary to produce and maintain a remission. Rarely has it been necessary to give more than 200 mg. daily during the first few days and this amount may be reduced greatly. In two patients with rheumatoid arthritis 25 mg. daily has been sufficient to maintain a reasonable satisfactory remission.

SUMMARY AND CONCLUSION

Corticotropin and cortisone have proven to be adjuncts in the treatment of collagen diseases. The use of these substances has produced no "cures" in any instance so far as we have been able to determine from a survey of the published case reports or from our experience. Undoubtedly, life has been prolonged in many instances and relative comfort has been given to patients for varying lengths of time.

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ADRENAL INSUFFICIENCY IN NEWBORN MALE INFANTS*

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The general awareness of pediatricians of insufficiency of the adrenal glands has been accentuated by several investigators with beautifully presented and well worked out cases of this disorder. However, the relative rarity of this syndrome makes a series of four such cases worth presenting. Adrenal disorders have been well classified by numerous investigators and it is unnecessary to give the classification again. Since the first such cases presented by Wilkins,[1] Talbot[2] and their co-workers, the literature has been gradually enlarged by case reports of other investigators.[3 4]

Some four years ago, such a case was presented by Chenoweth[5] at this medical meeting. Jaudon[6] has likewise presented a series of cases of temporary insufficiency of the adrenal glands. None of the cases presented here would seem to fit in with such a classification. The four infants to be described are all males and each presented very similar clinical picture and similar laboratory studies.

Case 1.—B. M. was a full term infant born January 15, 1948, of an uneventful pregnancy and normal labor. The birth weight was 7 lb. 9 oz. At ten days of age the baby developed severe vomiting and diarrhea and was admitted to the Baptist State Hospital. Parenteral fluids, both subcutaneously and intravenously, were administered and after five days he was discharged seemingly doing well. Three days later he was again seen and, without vomiting and diarrhea, he had become markedly dehydrated and presented an ashen cyanotic tint and was readmitted weighing 6 lb. 13 oz. Each admission presented only these findings, that is, dehydration and the ashen color. Again parenteral fluids, notably saline, plasma and blood transfusions were given, but the baby only maintained his weight and did not gain. Blood chemical analysis at this time revealed: nonprotein nitrogen 47 mg. per cent, total serum chloride 330 mg. per cent, calcium 13.2 mg. per cent and normal serum proteins. Hemograms and urinalysis were

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within normal limits. At that time serum sodium and serum potassium studies were unavailable at this hospital. We felt we were definitely dealing with an adrenal insufficiency. Adrenal cortical extract was administered 1 cc., every four hours with marked improvement and we were able to reduce the subcutaneous fluid administration substantially. Six days later desoxycorticosterone acetate (doca®) was begun, 1 mg. daily and this was gradually increased to 4 mg. daily. On added salt, 3 grams in the 24-hour formula, plus doca® and adrenal cortical extract, this baby gained satisfactorily and subcutaneous fluids were discontinued.

He was transferred to the Arkansas Children's Hospital March 21, 1948, where the above therapy was continued. It was noted that the penis seemed to be enlarging gradually and the testicles seemed to be quite small. Seventeen-ketosteroid determinations were obtained at this time and these were elevated to 3.25 mg. per 24-hour urine specimen (0-1 mg. being normal for this age). His state of hydration and weight gain continued with administration of doca® subcutaneously and the other therapeutic measures mentioned above.

On May 30, 1948, two pellets of doca® of 75 mg. each, were implanted subcutaneously. One week later the blood chemical findings were normal and the infant was discharged weighing 12 lb. 7 oz. The infant did well and gained excessively at home. However, the muscular development was rapid and much above that expected for his age. On March 28, 1949, he was readmitted to the Arkansas Children's Hospital and blood chemical studies revealed: chlorides 409 mg. per cent, nonprotein nitrogen 41 mg. per cent, serum sodium 230 mg. per cent and potassium 26 mg. per cent. The 17-ketosteroid determination was 3.25 mg. for a 24-hour specimen. Glucose tolerance test was performed and this revealed a low curve. On April 4, 1949, 2 pellets of 75 mg. each were again implanted subcutaneously.

On December 23, 1949, this child ingested twenty aspirin tablets of 5 grains each and the mother failed to seek medical attention for 24 hours. At this time, he was moribund due to acute salicylate intoxication and he died 2½ hours after admission to the hospital. Fortunately an autopsy was obtained. Grossly this presented a 23 months old infant with marked muscular development. The penis was unusually large and there was considerable pubic hair developing. Microscopic examination of the adrenals revealed "excessive development of the cortex with almost no medullary portions found. The zone of fasciculata is unusually prominent. The cytoplasm of the cortical cells in many areas appears to be disintegrating. Some of the periadrenal fat shows serous atrophy." Examination of the testes revealed "the tubules are small and not yet developed. The interstitial stroma shows very few Leydig cells. No spermatogenesis is found."

This patient died of an acute salicylate poisoning, the other three children of the series are today living and are described below.

Case 2.—T. P. was born full term an apparently normal male infant, weighing 8 lb. 8¼ oz. on March 31, 1949. He was discharged on the eighth day, gaining adequately on the formula. After discharge it was noted that the baby nursed poorly and a gradual weight loss occurred. No fever was present at any time and the baby had no abnormal stools or vomiting. On the seventeenth day, the baby was examined and found to be emaciated in appearance and extremely dehydrated. Hospitalization was advised. On admission he weighed 7 lb. 11 oz. Physical examination revealed only the dehydration and a rather ashen color. Laboratory studies revealed 4-plus albuminuria, hemoglobin 15.5 grams, a red blood count of 5,400,000 and a white count of 21,900. The differential presented 56 per cent polymorphonuclears, 36 per cent lymphocytes, and 8 per cent monocytes. At this time, with these findings, it was thought the infant had a urinary tract disorder and that the dehydration was on the basis of poor food and fluid intake.

Subcutaneous and intravenous glucose and saline were administered with prompt improvement. However, as the parenteral fluids were continued the baby persisted in weight loss and it was necessary to administer heroic amounts of parenteral fluids to maintain the body weight. Four days after admission the urinary findings had returned to normal, but despite this the baby lost to 7 lb. body weight. On the fourth hospital day the baby was receiving approximately 20 per cent of his body weight in parenteral fluids daily without gaining weight. Blood chemical study at this time revealed: nonprotein nitrogen 45.5 mg. per cent, creatine 1.7 mg. per cent, blood chlorides 460 mg., serum potassium 23.5 mg. per cent and the serum sodium 305 mg. per cent. We felt we were definitely dealing with an adrenal insufficiency at this time. Doca® was started, 2 mg. intramuscularly daily, on the fifth day of hospital admission and was gradually increased to 6 mg. daily. Cortical extract 1 cc. every eight hours was administered simultaneously and 3 grams of sodium chloride was added to the formula. The baby immediately began to gain weight rapidly and the ashen color disappeared. The appetite and general appearance improved remarkably. All subcutaneous fluids were discontinued on the eighteenth hospital day. By adjusting the dosage of doca®, decreasing the adrenal cortical extract and decreasing the added salt to the formula, we established a baseline of 2 mg. of doca®, once daily and 1.5 grams of added salt to the formula. On this regime the baby did well and gained at about the expected rate. On the sixtieth hospital day the infant was discharged, to be handled as an office case in so far as treatment was concerned. Subsequently this infant was given injections of doca®, 2 mg. daily, hoping that this would be one of the incidence of temporary adrenal insufficiency reported by Jaudon. Doca® was continued 2 mg. daily until July 21, 1949, at which time it was entirely eliminated. The weight was noted to fluctuate up and down, but by increasing or decreasing the salt we were able to maintain a rather steady weight gain and this continued with an occasional dose of doca® until October 31, 1949, at which time he was admitted to the hospital and two pellets of doca® were implanted subcutaneously (75 mg. each). His condition remained satisfactory and at the end of one year, he weighed 22 lbs. 1 oz. and was doing quite well. At times the added salt was increased or decreased, depending on the weight gain or loss

as manifested. On one or two occasions he lost as much as 20 oz. in a period of 48 hours and by increasing the salt he quickly regained this weight. This condition persisted up until November of 1950, at which time two more pellets of 75 mg. were implanted subcutaneously. He had gone 13 months between implantations of doca®.

At the age of 2 his weight was 31 lb. 6 oz., at which time the family moved to South Carolina. He was followed in this state by another pediatrician and his weight gain continued, being 36 lb. and height 38½ inches at the age of 3 years.

In February of 1952, while visiting in Arkansas, this child developed a severe diarrhea and vomiting and was hospitalized. Despite rather large amounts of subcutaneous saline the condition progressed downward and he was again referred to us. Serum potassium was 28 mg. per cent, sodium 247 mg. per cent, the chlorides (after parenteral sodium chloride) were 561.2 mg. per cent. A 17-ketosteroid determination revealed 11.8 mg. per cent output for 24 hours, but blood chemistry had remained normal. At this time he was given doca® subcutaneously, parenteral fluids, and plasma, and the blood chemical findings quickly returned to normal. Again two pellets of 75 mg. each of doca® were implanted and after a week's hospitalization he was discharged. At the present time the family has returned to South Carolina and the patient is now under the care of a pediatrician with whom we had recent correspondence. The child now has a 17-ketosteroid urinary excretion of 11.65 mg. per 24-hour specimen. Cortisone has been added in the effort to decrease the androgen excretion.

Case 3.—L. K. was first seen at the age of six weeks, July 27, 1949. He was a full term infant the result of a normal pregnancy and labor. Birth weight was reported at 9 lb. 6 oz. and at 6 weeks the weight had decreased to six pounds and 2 ounces. At the age of four weeks vomiting and diarrhea had developed and despite numerous formula changes, this continued until the child was referred to us. On physical examination he was acutely ill, with a temperature of 102.6 and he was markedly dehydrated. No other abnormalities were noted. Laboratory studies revealed a red count of 3,930,000, hemoglobin 12 grams, white cells 15,350 and the differential showed nothing abnormal. Urinalysis revealed a 2-plus albuminuria with no other abnormal findings. Blood chlorides at this time were 526 mg. (the child had had subcutaneous saline), and the nonprotein nitrogen was 35 mg. He remained in the hospital for 7 weeks, being treated with oxygen, subcutaneous fluids, and antibiotics. On the second hospital day, cyanosis developed and an x-ray of the lungs showed a small area of consolidation in each lower lobe. This cleared satisfactorily on the antibiotics and oxygen therapy. He gained slowly with added salt and subcutaneous fluids. On the eleventh day the serum sodium was found to be 300 mg. per cent; potassium was not obtainable.

On the fifteenth day doca® was started with almost immediate improvement. Subcutaneous fluids were stopped, weight gain was steady. Eventually we reached 4 mg. of doca® daily and the weight gain progressed steadily to 9 lb. 14 oz. on the fifty-fourth hospital day. The infant was discharged with the referring physicians instructed to give 2 mg. of doca® daily. After 10 days he was readmitted to the Arkansas Children's Hospital. At this time he weighed 10 lb. 1 oz. Laboratory studies again revealed nonprotein nitrogen of 52 mg. per cent, serum sodium of 285, potassium 27.8 and 17-ketosteroids of 0.765 mg., which was interpreted as normal to slightly elevated. On doca® the child continued to gain and do well. Extensive laboratory studies were carried out and a repetition of the 17-ketosteroid determination showed 1.184 mg. excretion for 24 hours.

This child was maintained on added salt and doca® 3 mg. daily and gained slowly to 16 lb. 1 oz. On January 1, 1950, two 75 mg. pellets of doca® were implanted subcutaneously and the child was discharged on January 11, 1950. Subsequently he has gained, has become muscular, developed a moderately enlarged penis, but the testicles remain small. However, no subcutaneous pellets have been implanted since we have had considerable difficulty in following this child and getting the parents to cooperate in bringing him back for follow-up studies.

Case 4.—R. P., a two months old infant, was first seen December 2, 1949, with a history of loss of weight, vomiting, and diarrhea. The birth weight was 7 lb. 8 oz. and the weight at two months of age was 6 lb. 1 oz. The skin was pale and darkly pigmented and the child was markedly dehydrated. A definite state of shock was present. In addition carpopedal spasm was noted. He was admitted to St. Vincent's Infirmary with a diagnosis of pneumonia, tetany, and malnutrition. With oxygen therapy, antibiotics and calcium, plus parenteral fluids he improved rapidly and was discharged weighing 7½ lb. He did well until three weeks later at which time he was again seen with marked dehydration and in a state of shock. He weighed 6 lb. 3 oz. at this time. He was rehospitalized and again treated with parenteral fluids but failed to progress satisfactorily. He was then transferred to Arkansas Children's Hospital. Blood chemical studies revealed serum sodium of 300 mg. per cent and potassium of 20 mg. per cent. Blood chlorides were 495 mg. per cent, nonprotein nitrogen was 50, and creatine 1.35 mg. The Wassermann was negative and chest x-rays were normal. Seventeen-ketosteroids were 1.38 mg. per 24-hour urine excretion. He was given added salt in his formula, parenteral saline and doca® of 2 mg. daily, on which he was maintained quite well, eventually being able to maintain a constant weight gain without parenteral fluids. He gained steadily until March 19, at which time he weighed 12 lb. 4 .oz. One pellet of 75 mg. doca® was implanted subcutaneously on February 20, 1950.

He was readmitted March 31, 1950, with a gastroenteritis and responded well to subcutaneous saline administration. On April 3, 1950, he was discharged with added salt in the formula. He was seen in our office May 4, 1950 and was progressing well, weighing 14 lb. 15 oz.

The family disappeared and this child was not again seen until October 24, 1952. Except for added dietary salt and an occasional craving for salt, he had done well and was quite muscular, though not exceptionally large. He had had no severe illness. This child comes from a family of small body build and it is worth noting that he is larger and weighs

some 6 pounds more than a brother eighteen months older.

DISCUSSION

As pointed out in the case histories of these infants, certain similarities existed in all four cases. Among these were the absence of abnormalities early in the course of the disease other than dehydration and the presence of the state of shock in the earlier stages or in crises. In addition, two of these cases were in small children four to eight weeks of age with the same presenting disease, pneumonia. The other two cases each had diarrhea and vomiting as presenting complaints. Only one patient showed pigmentation of the skin and mucous membranes. Difficulty in maintaining water balance was an outstanding feature in all of these cases. Marked anorexia, listlessness and failure to gain weight, even loss of weight, despite huge amounts of parenteral fluids was an outstanding finding. Diarrhea that developed in two of these cases was of a type unaided by the usual therapeutic measures. Infections were present in two of these children, which seemed to accentuate the development of the syndrome as two of the cases referred to above had pneumonia. One of the children exhibited moderate hypoglycemia and a glucose tolerance test presented a low curve.

Extensive other laboratory procedures were carried out, such as blood cultures, x-ray studies, electrocardiographic studies, serum protein determinations, cholesterol, studies of calcium and phosphorus metabolism, all with normal findings.

It has been pointed out that a high familial incidence occurs in these cases, but this was not observed in any of the families of these children. *Case 1* was the first pregnancy, but two subsequent pregnancies have resulted in apparently normal children. *Case 2* was an only child, with a history of a miscarriage early in pregnancy prior to the birth of this child. *Case 3* was the sixth pregnancy, none of the other siblings exhibiting any abnormalities which we could observe. *Case 4* was the third pregnancy, the first two being normal and subsequently two other pregnancies have developed, neither of which has showed this type of disturbance.

The treatment of each of these cases initially was the correction of shock and reestablishment of fluid balance. Infections present in two of the infants when first seen were treated with antibiotics, plasma, whole blood transfusions and oxygen after correcting the initial shock and dehydration. As soon as the diagnosis was confirmed by blood chemical studies, added dietary sodium chloride and intramuscular adrenal cortical extract were given. When the fluid balance was stabilized doca® was begun and increased gradually until all subcutaneous fluids were discontinued. Then attempts to determine a maintenance dose of doca® were made on the basis of weight progress. After the maintenance dose of doca® was determined the infants were treated for varying intervals by the intramuscular route. Subsequently pellets of doca® were implanted in all of these infants. The interval between pellet implantations has varied. The two first children have each had several pellet implantations; the third child had one pellet implantation and has since been maintained on a high salt intake. The fourth child after the initial implantation, was not seen for 2½ years and only recently were we fortunate enough to see this child again. The mother notes, however, at the present time, that he requires considerable salt with his food and has sometimes been seen to seek excess salt on his own. Except in crises none of the infants required doca® intramuscularly between implantations. The indications to determine when other pellet replants were necessary were based on weight loss and deviation from normal of blood chemical findings.

As will be noted in the cases presented, two of the infants had parenteral fluid therapy prior to admission. In these the initial blood chemical findings were not appreciably abnormal. Yet, later studies revealed the typical changes consistent with adrenal insufficiency. In all of these infants at some time in their course the blood serum potassium was elevated, serum sodium decreased, blood chlorides decreased, nonprotein nitrogen elevated and in all cases the excretion of 17-ketosteroids was increased. In the series of four cases it will be noted that both the excessive androgen excretion and addisonian-like crises occurred.

In general, since the initial treatment, all of these children except *Case 2*, which died of salicylate poisoning, have been remarkably free from infections. All are larger than

normal and present varying stages of excessive muscular build. The first two have large penile development, with small undeveloped testes. *Case 2* on autopsy at the age of 2½ years, showed pubic hair developing and absence of spermatogenesis, with poor interstitial development of the testes. In *Case 1*, ossification studies done at the age of 16 months showed a bone age normal for the chronological age.

SUMMARY

Four male infants with adrenal insufficiency have been presented. These cases have clinically shown difficulty in maintaining water balance shortly after birth. All these infants had abnormal blood chemical findings, namely: elevated nonprotein nitrogen, elevated serum potassium, decreased serum sodium and decreased blood chlorides. In addition, each infant had an excess androgen excretion. Desoxycorticosterone acetate pellets were implanted in each. Excessive muscular development and growth of the penis were noted in all of these infants.

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PHEOCHROMOCYTOMA*

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The adrenal glands anatomically consist of two parts, the medulla and the cortex. It would appear that any further interdependence ceases to exist. The adrenals may be likened to the posterior and anterior portions of the pituitary or to the thyroid and parathyroid glands which have close physical proximity but possess different sources of origin, different structure and different functions.

Pheochromocytomas are tumors which arise from cells of the adrenal medulla. Identical, hyperfunctioning neoplasms originating in the extra-medullary chromaffin tissue are frequently called "paragangliomas." In all patients suffering from hypertension, be it paroxysmal or sustained, the possibility should at least be entertained that they possess a pheochromocytoma. It has been only during the past twenty-five years that the surgical removal of these tumors has been accomplished. At present, according to Roth *et alii*[1] about 150 operations for the removal of these lesions have been recorded in the literature. From this, one may be lead to believe their occurrence is extremely rare. This, however, does not appear to be the case. Graham,[2] in his collective review and analysis of 207 cases in 1951, points out that of 1,700 unselected patients undergoing lumbodorsal splanchnicectomy for hypertension, 8 unsuspected pheochromocytomas were encountered, an incidence of almost 0.5 per cent. He speculates that since 175,000 persons die each year as the result of hypertension, about 800 of these may be due to an adrenal medullary tumor. DeCourcy[3] has emphasized that in the so-called malignant hypertension group, the percentage of pheochromocytomas may be 5 per cent or higher. Of 31 of their patients undergoing bilateral subtotal adrenalectomy for malignant hypertension, three were found to have chromaffin tumors. Berkheiser and Rappaport[4] reported finding, in a general hospital over a six-year period, five unsuspected pheochromocytomas in 2,012 consecutive autopsies, an incidence of 0.25 per cent. At the George Washington University Hospital and the Walter Reed General Hospital, we have not had a similar experience during the past six years. Evidence has shown that patients with hypertension resulting from pheochromocytomas may anticipate rather dramatic and usually permanent relief following the surgical removal of the tumor. Dr. C. H. Mayo[5] removed a tumor in 1927 which was shown to be a pheochromocytoma; however,

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the first patient in whom a diagnosis of pheochromocytoma was made preoperatively was operated upon by Dr. Arthur Shipley[6] in 1929. A long follow-up of this patient, over the years, proved that the removal of the tumor gave complete and permanent relief from her attacks of paroxysmal hypertension.

An additional long term follow-up has recently been obtained on the patient reported in 1934 by Coller, Field and Durant.[7] In August 1952, twenty-one years following the removal of a pheochromocytoma for paroxysmal hypertension it was learned that the patient had had no attacks since his operation and that renal, cardiac, and blood pressure studies were all normal. Additional long term studies will aid in promoting the already prevalent idea that hypertension resulting from pheochromocytoma can be cured surgically. This alone should stimulate great interest in all who deal with hypertensive patients to be on the alert and search for these neoplasms as a possible cause. In this way it may become possible to reduce the number of needless hypertensive deaths occurring each year as a result of these tumors.

In cases of episodic hypertensive attacks occurring in normotensive individuals, the possibility of the existence of a pheochromocytoma may be considered rather early. On the other hand when the less dramatic but no less frequent manifestation, namely, sustained hypertension, results from activity of these tumors, there is usually much time lost before their presence may be considered, or sought for.

Since chromaffin tumors can cause hypertension, and since this condition can be relieved by their removal, it seems paramount that an extensive search be made for those few patients to whom a surgical cure may be offered. Such a course is not without great difficulties. The diagnosis of a pheochromocytoma may be extremely difficult and the mortality of 20 to 25 per cent associated with surgical removal remains too high. Overcoming these obstacles requires optimum teamwork between internist and surgeon and also various aids which can be used, not only to establish a diagnosis, but to screen all hypertensive patients.

For the two types of hypertension due to pheochromocytomas there are two varieties of drugs to aid in making a diagnosis. In those individuals who suffer from paroxysmal attacks, histamine, tetraethylammonium chloride, and mecholyl, are utilized to create a characteristic attack. The employment of these drugs is based on the premise that their administration to patients with paroxysmal attacks of high blood pressure will precipitate a characteristic attack if a pheochromocytoma is present.

To aid in the diagnosis of sustained hypertension due to hyperfunctioning adrenal medullary tumor, piperoxan® (benzodioxan 933-F,® benodaine®), dibenamine® and C-7337 regitine® have been utilized. Their value is based on their ability to block the adrenergic effect of the tumors, and this inhibitory effect results in an immediate drop in blood pressure that is of diagnostic significance. Because of the effects of extrinsic influences, patients undergoing any of these tests should be carefully prepared. Individual variations in patient response require skillful interpretation of the tests. False positives or false negatives may occur and require repetition of the test in order to obtain a more accurate appraisal.[8-13]

The sympatholytic or adrenolytic drugs which counteract the sympatho-adrenal epinephrine or norepinephrine effects have other than diagnostic value. They may be utilized to control attacks in the paroxysmal type, they may be employed to counteract hypertension during the work-up stage, and finally they have a real use in the preoperative preparation and during the operative procedure.

In addition to the drugs that may throw light on the diagnosis, additional information may be obtained by a thorough physical examination and an extremely careful history. During abdominal palpation, a tumor may possibly be felt or a paroxysmal attack may be initiated.

X-ray studies may reveal a soft tissue mass on an abdominal scout film. An intravenous pyelogram may reveal downward displacement of the kidney on the affected side or a soft tissue shadow in the region of the adrenal. Kaplan and Green[14] studied 72 excretory urograms in patients with clinical evidence of suprarenal tumors. Of 39 patients from whom tumors were subsequently removed, 29 tumors were demonstrable. In the remaining 10, no abnormality could be demonstrated. In 1929, Cardelli[15] developed a

technic for radiographic visualization of the kidney outline by injecting air into the perirenal space. This method did not receive much attention until 1935 when Cahill[16 17] popularized the procedure. We have not employed this technic in hyperfunctioning medullary tumors because of mishaps that may occur. In 1950 Rivas[18] described a method of presacral retroperitoneal oxygen insufflation. We have found this to be an effectual and easy way to outline the retroperitoneal structures.

Steinback, Lyon, Smith, and Miller[19] have just reported that there have been no fatal complications in a series of 1500 collected cases. This apparent element of safety is most encouraging. The method consists of introducing a No. 18 spinal needle into the space between the rectum and sacrum through a skin wheal in the perineum. Under no more than 15 to 20 cm. of water pressure 15 cc. of oxygen per kilogram of body weight is injected.

Once the diagnosis of a hyperfunctioning chromaffin tumor has been established or even strongly suspected, surgical intervention becomes advisable. Preparation of the patient by the internist is begun which now usually includes the administration of one of the antiadrenergic drugs. This is done in an attempt to prevent the occasional hypertensive crisis that may occur at operation during the manipulation of the tumor. During the operation the internist and anesthesiologist closely cooperate in any further administration of adrenolytic drugs or in the use of vasopressor agents to combat any extreme circulatory collapse. The anesthesiologist has the additional problem of providing adequate relaxation of the patient. It is the duty of the surgeon, as the third member of the team, to remove the tumor as quickly, as carefully, and as gently as possible.

The upper abdominal transverse incision has two definite advantages over other surgical approaches. It permits simultaneous exposure of both adrenals because in about 10 per cent of cases bilateral tumors may be encountered. It also allows exploration of the periaortic regions and organ of Zuckerkandl as about 10 per cent of the chromaffin tumors have an extra-adrenal location. It may also have an additional advantage in that the patient is in a suppine position and can easily be placed in a Trendelenburg position. Bartels and Cattell[20] have said that the hypotensive state with collapse and death which may follow the removal of a pheochromocytoma is due to acute left heart failure resulting from epinephrine intoxication and should be treated by the Trendelenburg position and no vasopressor drugs are necessary. Possibly the preparation of patients with adrenolytic drugs will confirm this impression by preventing the epinephrine excess and secondary hypotension, but at present we agree with Priestly,[21] Richards and Hatch,[22] and others that the use of vasopressors seems justifiable and necessary.

We have considered that the first 48 postoperative hours constitute a critical period. Any difficulty encountered in the early hours of this period may be due to medullary insufficiency. Tumor hyperfunction has suppressed normal cell activity and some time may elapse before these cells resume their function. After the first few hours, adrenal cortical insufficiency may become a problem. It is felt that this cortical disturbance may result from the trauma of manipulation, exploration and removal of the tumor. During the critical period, vasopressors and adrenal cortical extract preparations in adequate quantities are employed along with general postoperative measures.

During the past 18 months, we have had three hypertensive patients who gave positive reactions to the test drugs which indicate the presence of a chromaffin tumor. These are being reported in detail elsewhere, but are mentioned here to emphasize certain points.

Two of these patients were women. One was a 29-year-old housewife who was normotensive until the seventh month of her first pregnancy in January 1951. At that time because of some supposedly allergic headaches, histamine was administered to the patient. This was immediately followed by an attack which, she was informed, was due to nervousness. During the ninth month, she had another episode which occurred spontaneously and simulated the first attack. At term, she was delivered of a living child and promptly went into profound shock, but recovered. She continued to have these attacks and was first seen by us in May 1951. Tests and x-rays indicated a pheochromocytoma in the left adrenal gland. The tumor was removed, and the abdomen explored through an upper transverse abdominal incision on May 18, 1951. At this writing, the patient has remained symptom-free and in excellent health.

The other woman patient was 31 years of age and

had formerly been a nurse in the Army, during which time her blood pressure was known to have been normal. In 1947, during the eighth month of her first pregnancy, she was told that her blood pressure had become so elevated that labor must be induced prior to her expected date of confinement. After delivery, the hypertension apparently subsided and the pressure remained normal until the eighth month of her second pregnancy in late 1948 at which time it again became markedly elevated, and remained so until after delivery. Once more it returned to normal. In 1950, during her third pregnancy she began to be hypertensive again (200/120) and continued to remain so when she was first seen by us on July 24, 1952. At this time, she presented the picture of a rather severe malignant hypertension. During the work-up, it was discovered by x-rays that a tumor was present in the left adrenal gland and tests were positive for pheochromocytoma. On August 19, 1952, the abdomen was opened through an upper transverse incision. After careful exploration, the left adrenal tumor was removed. The patient promptly became normotensive and has remained so to date. The cardiovascular renal changes as well as the symptoms that resulted from the prolonged hypertensive state have almost completely disappeared during the past three months.

It is interesting that the first patient's original attack was precipitated by one of our test drugs, histamine, which was used as a therapeutic agent for what was thought to be an allergic cephalalgia. In the second case, it is interesting to speculate that had the antiandrenergic drugs been employed earlier to screen her hypertension, a diagnosis might have been arrived at much sooner, thus sparing her three occasions of being considered a toxemia of pregnancy, as well as the sequelae resulting from a prolonged hypertension.

The third patient was a 22-year-old white man. He had apparently been quite healthy until November 1949. He had been a football player, but had to discontinue the sport because of substernal pain, headaches and the physical finding of hypertension (185/120). His symptoms and elevated pressure persisted for almost two years and were present when first seen by us. X-ray studies failed to reveal any abnormality. However, the tests using adrenergic blocking agents persistently indicated the presence of a hyperfunctioning chromaffin tumor. In our opinion, this finding warranted surgical exploration, which was carried out through a transverse upper abdominal incision on September 10, 1951. No tumor could be found and a biopsy of each suprarenal gland revealed normal tissue. The postoperative course was uneventful. Following operation, the tests continued to indicate strongly that the hypertension was due to a chromaffin tumor, possibly located in the thoracic region since tissues exist there that might give rise to such a neoplasm. The problem was presented to the patient, and an exploratory thoracotomy advised. To date this has been refused.

This particular case is mentioned merely to emphasize that the diagnostic tests are extremely reliable, and when repeatedly positive should cause continued search for the tumor even if it requires multiple operative procedures.

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RESULTS OF THREE YEARS EXPERIENCE WITH A NEW CONCEPT OF BABY FEEDING*

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During the past three years I have been employing in my practice a baby feeding schedule which revolves about a 6-hour feeding interval from birth with the introduction

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of solid foods as early as the second day of life. The story of the origin of such a revolutionary schedule along with the *raison d'etre*, its details, advantages and results will be the subject of my paper today.

The reading in one of the medical journals in 1942 a paper which emanated from New Orleans, so exaggerated my dissatisfaction with the manner in which I had been taught to feed babies that I shortly began to deviate from the milk-for-five-months diet to one marked by the early introduction of solid foods. Consequently, I found myself putting infants of three to four months on a three-meal-a-day diet. Inquiry among pediatricians of recognized ability in my locality as to what was bad about such an idea (having already been convinced as to what was good about it) brought forth the very unscientific reply, "We do not think their little stomachs are big enough to hold sufficient food at this age to carry them through." Strangely these same pediatricians have long since adopted these then seemingly radical ideas.

A continued pursuance of a practice, general in every sense of the word, gradually brought me to the realization that many of the present day physical and functional disorders seen, are directly due to the tension and fears of present day living. A conviction that many modern mothers fall into this category, mainly because of their undue concern as to the welfare of their babies, plus the fact that they are always tired and fatigued as a result of constant care for the demands of their babies, with the resultant loss of sleep and rest, started me to thinking how such a situation might be remedied. Naturally in general practice there are many babies with minor illnesses that are of little import, or with behavior and feeding problems. I was impressed by the profound disturbance of mothers over these minor problems, and through a gradually evolutionary process of thought on these matters I became convinced that we physicians lay too much stress on the baby's welfare and pay too little attention to the effect of these matters on the mother. I wondered whether one could not discover a feeding method, or should I say a baby care routine, which would take the stress off the mother, would simplify her obligations towards the baby and still produce a healthy, contented infant. A study of infant feeding procedures through the ages and in different geographical locations was impressive in one common denominator: the continued healthy survival of infants despite what feeding practices were employed. The natural process was then to hit upon one that, without jeopardizing the baby's health in any way, would be less demanding on the mother's time, patience and good nature. The process of these ruminations led me into many contiguous fields which had to do with customs and procedures involved in baby care all of which resulted in the firm conviction that it is rather difficult to find anything that will hurt a normal newborn. These mental gyrations were all part of a subconscious process involving years of baby care on my part and with it the stress I laid on babies became less and less and at the same time their feeding routines became more and more simple. It is my feeling that when the mother manifests signs of over attention, indecision and insecurity the baby in time becomes affected by these manifestations. In other words, to me an unstable mother will insure an unstable baby.

An expression of my ideas on baby feeding to a receptive obstetrical patient, the mother of four other children, resulted in her asking following delivery, that her infant be put on three meals a day. To my delight and that of the nurses the idea worked to a degree far exceeding expectations. The second infant to be put on a three-meal-a-day schedule was a 5 pound 12 ounce infant which I had the good fortune to follow daily because of a continued hospitalization period, resulting not from any physical ailment but because the baby was up for adoption. Again I was pleased by the very normal neonatal course this baby followed while on this new schedule.

As I continued to put more and more babies on this routine (at first all were the babies of volunteer mothers) I found I had many critics in the guise of grandparents, well meaning friends and neighbors, particularly those of the apartment house variety. In order to placate some of the neighbors, relatives and apartment house dwellers who could understand and condone the crying of a baby fed frequently but not that of an infrequently fed baby, a midnight feeding was added to the three-meal-a-day schedule, thus making it a 6-hour interval schedule.

At this time, all babies under my care are on this 6-hour schedule. I would estimate that 300 babies have been and are being raised on this new regimen. I first introduce my new obstetrical patients to my baby feeding ideas at one of their early prenatal visits. I encourage them to observe these babies as the opportunity presents itself in my waiting room and to feel free to question the mothers of the babies on the 6-hour schedule.

At the time I discharge my obstetrical patients from the hospital I spend quite a little time, either singly or in groups, going over in great detail the favorable points of this schedule. I first impress upon them the idea that this is truly a philosophy of baby feeding rather than any new idea or system. I often admit that I have only borrowed from the mother with 8, 10, or 12 children her ideas of baby feeding, altered as they are by her restricted time, and polished them up a little for the modern mother with her much smaller family. I try to impress these mothers with the idea of training the baby to fit into their lives rather than embarking on a life in which they are constantly and frequently altering the course of their own lives to fit into the baby's life. I admit readily these babies will cry, but steadily maintain that at no time any more than other babies, with the added assurance that shortly the amount of crying will lessen as the baby learns that the crying gains him naught. Indeed, in my experience in private practice along with that of 6 years of caring for institutional babies I am sincere in my belief that the normal baby will fit into any given routine; it is the mother who has trouble fitting into a routine.

In instructing the mothers I then enter into the details of the schedule impressing upon them that this is a strict 6-hour feeding interval schedule whether it be at the suggested 6, 12, 6, 12 or one of their own choosing such as 8, 2, 8, 2. It might be well here to outline the essential details of the feeding routine which I use but which could be adapted to the individual variations of any physician *(Table 1)*.

Schedules are discussed in general. All foods are started with one teaspoon amounts. I admonish these mothers to continue to awaken their babies for the midnight feeding until they themselves are ready to drop that feeding regardless of the protestations of the baby. While many mothers do it before, I suggest a time of 17 days for dropping this midnight feeding and give them signs, namely the difficulty of awakening and the diminishing amount taken at that feeding as criteria of the baby's readiness for such an omission. Moreover, I warn them, if they go into this phase of dropping the midnight bottle with the mental reservation of giving a bottle if the baby cries, not to drop this feeding but to continue it at its regular time. However, once this feeding is dropped it is never to be resumed.

Once these babies are on a three-meal-a-day routine, I then suggest to the patient that these babies are no longer on a strict schedule but are eating three meals a day; however, at times chosen for the mother's convenience and not at the baby's insistence. It is my urging that by 3 to 4 months these babies will be taking breakfast anywhere from 7 to 9 in the morning, lunch at anywhere from 12 to 2, and dinner anywhere from 5 to 7 in the evening, indeed a flexible and most desirable routine to most mothers. I stress to these mothers that I want them to be independent mothers with only a minimal amount of guidance from me. It is my practice to see these normal babies formally only twice in the first year, once at the time I give the mother her 6 weeks postpartum check and again at the 5 to 6 months period for checking and the start of their immunization injections. On each of these occasions

SIX-HOUR FEEDING SCHEDULE

Given Mothers at Time of Leaving Hospital

Feedings at 6:00 a.m., 12:00 noon, 6:00 p.m. and 12:00 midnight. Water between feedings.

At 2-3 days of age: Cereal at 6:00 a.m. and 6:00 p.m. (Oatmeal and barley suggested as starters.)

At 10 days of age: Strained vegetables at 12:00 noon. (Peas, beans, carrots suggested as starters.)

At 14 days of age: Concentrated cod liver oil, 2 drops a day to start. Increase one drop a month up to 5 drops daily.

At 17 days of age: Strained fruits at 6:00 p.m. (Applesauce, peaches, pears suggested as starters. May decrease cereal.)

Midnight feeding may be dropped at any time.

At weekly intervals add:
3 weeks—Orange juice and sterile water, equal parts of each, up to 2 ounces of each at 6:00 a.m.
4 weeks—Strained meats
5 weeks—Custards
6 weeks—Soups
7 weeks—Mashed banana
8 weeks—Egg

Formula as indicated.

TABLE 1

weight and hemoglobins are taken. In addition to these visits the mothers are encouraged to bring their babies in so that my nurses may weigh them and get hemoglobins on them frequently. These mothers are also encouraged to call one of the nurses in my office who is completely familiar with this routine, if any questions arise on which they are in doubt.

Individual charts, in addition to the usual office records are kept on these babies showing weight increments and blood levels. From these charts I have attempted to draw conclusions as to weights and blood trends. The weight would seem to follow a pattern with a lesser initial weight loss, a slower primary rise and finally a delayed but definite rise to average levels. The babies on this schedule have represented a wide variation in initial weight. The smallest baby was 4 pounds 12 ounces at birth but quite normal. The largest was well over 10 pounds. Blood levels seem to follow those on control regimes. For our subtropical Miami clime no blood standards have been set up so our work on this score is handicapped. Regardless of the use or nonuse of vitamins and mineral preparations these findings seem to undergo wide fluctuations. I recently saw a 5-months-old baby with a hemoglobin of 76 per cent which represented a rise of 26 points from 52 per cent at 2 months, on no hematinic therapy whatever.

RESULTS

Whereas all this may sound wonderful on paper the inevitable question arising is one concerning results. Unfortunately it is impossible to reduce to statistics the constant flow of favorable comments and enthusiasm which is forthcoming from these mothers both directly and indirectly. However, blood levels and weight charting seem to be the main concern of those who set the standards for evaluating babies. A routine check on these aspects would tend to prove that these 6-hour babies follow closely the physical standards set up by control babies, namely those on a more conventional type of feeding regime. In a preliminary survey on well over 100 babies, overwhelmingly favorable reports were given on observations as to time of sitting, standing and walking, appearance of first tooth, on questions as to ease of management, incidence of bowel upsets and allergies and the ease of new food introduction. One of the most startling results to come out of this preliminary survey was the finding that 99 per cent were able to follow the early introduction of foods closely or moderately well. It is probably this phase of the regime which has the greatest popular appeal and approbation but it remains my observation that the acceptance of this phase spells for success of the 6-hour phase. It is my feeling that the two phases of this program are reciprocals one of the other in determining the success of the whole program.

At a long time glance it is my feeling that these babies as they continue to grow will present less in the way of feeding problems particularly if the parents will continue to follow the principles of this regimen. It has been and always will be my insistence that hunger is the best, but most commonly neglected, tonic in the presence of feeding problems in infants and children.

The majority of mothers with babies on this schedule are wont to maintain that the infants are healthier, faster developing babies, but naturally I am reluctant to accept this plaudit. I am content to maintain that they are just as healthy.

The criticism of this concept centers about four main points, namely: (1) question of nursing, (2) frequency of allergies, (3) question of the baby's ability to swallow and assimilate these foods, and (4) allegation of the omission of the opportunity to manifest parental love. These are all well taken but can easily be answered and in my opinion all refuted.

On the question of nursing I find my percentage of mothers nursing successfully quite low, but still no lower than it was when I was using a 4-hour schedule. The mothers who nurse have no more difficulty going 6 hours than my nursing mothers during the war who often nursed only morning and evening on the resumption of their wartime jobs, this often as early as 3 to 4 weeks following delivery.

If one is to accept the new basic concepts of allergies, particularly as viewed by Rinkle one cannot help deciding that with the early introduction and rotation of foods there should be fewer allergies. Indeed what could have more allergenic potentialities than a diet restricted for months to one single food sub-

stance such as milk, other than the mother's own milk.

As to the question of swallowing solid foods, I find absolutely no inability on the part of mothers who feed solids from a spoon when started practically at birth. This is in direct contrast to the troubles my mothers used to have when waiting until 6 or 8 weeks to start solids. On the question of the assimilation of these foods I found a willing and most progressive ally in the guise of one of the baby food houses who produced scientific article after scientific article, all favorable, when asked the question concerning what work would have to be done to prove ability to assimilate foods on the part of these babies.

It is not my intention to omit any opportunity for the manifestation of the parental instincts. Rather it is my desire to advise more wisely the time for such manifestations. I urge my patients to fondle, cuddle, love and rock their babies, but only when they are happy and not when they are crying. It is my own feeling, the child psychiatrist notwithstanding, that by granting a child what it wants when it cries, be it only food, that we are fostering a definite pattern of demand which will be continued throughout childhood.

The applicability of such a routine has far exceeded my narrow field of visions. I have had a number of requests for details of this idea from countries where milk is not readily available, namely West Africa, the Dominican Republic, and Paraguay. These countries, primitive as we may picture them to be, still encounter the problem of insufficient lactation on the part of the mother. At the present time the governments of the Dominican Republic and Paraguay are having the original article translated into Spanish for distribution throughout the maternal and well baby centers in the hope that with the early introduction of foods the infants getting insufficient breast milk can be started on solids rather than depend upon the importation of expensive canned or powdered milks for supplementary feedings. You may be sure I shall follow this work with great interest.

In summary I might say that use of a baby feeding regimen that is comprised of a 6-hour feeding interval which is rapidly converted to a three-meal-a-day regimen, along with the introduction of food other than milk as early as the second day of life continues to be a source of gratification to its users and also a source of medical interest as these babies continue to follow closely and sometimes even surpass the physical standards set up by babies on a more conservative routine. A more detailed survey, now in progress, which will cover some 300 babies would seem to emphasize the results of our preliminary survey done over one year ago. In addition these results would seem to be equally satisfactory in babies not under my direct supervision as the schedule reaches out to friends and relatives in my own locality, in other states and in foreign countries.

DISCUSSION *(Abstract)*

Dr. Benjamin M. Cole, Orlando, Fla.—With Dr. Sackett's 300 personal case records, and because many of us with a smaller series are just as enthusiastic, I am convinced that he has a concept that is sound and logical and that is gaining supporters everywhere.

As I listened to him today and as I have reviewed his past work, it seems to me that there are several pertinent points to be brought out. First is the fundamental philosophy of this new concept. It has been our custom in the past to remind the family that a frail new baby was coming and that they were to conform to a whole new schedule of living when it arrived. The father was to feed it at night so mother would be fresh and rested for the constant daily routine. In the daytime other members of the household had to be quiet so baby could sleep or they must postpone their meals so baby could eat exactly on time.

To me the idea of having a baby conform to the family routine, rather than having the whole family conform to the baby is a wholesome, stable thing to do. It contributes to the stability of the family, the mother and the baby.

Second, the very early supplementary feeding of solid food has many merits. We see many new mothers who have insufficient milk early. The milk just does not come down as it is supposed to do in sufficient quantity to keep baby full. The mother puts him to breast often to stimulate the flow and stop the hungry child from crying. Often times during these early days there is no increase and mother winds up instead with sore, cracked nipples and baby is given a supplementary bottle. He learns immediately that he gets more milk and gets it more easily this way and the result is another mother who cannot nurse her baby. By offering solid food early we can dispense with this early bottle, keep baby satisfied and spare mother's breasts until she produces milk and adjusts to nursing. By the same token we see bottle babies who will not take enough milk at a nursing to last to the next feeding. They are just not interested in sucking. Some solid food in their stomachs will carry them much longer. Third, there may be those who ask how you can teach a newborn to eat. To this I reply that they learn faster than at any other age and offer the follow-

ing evidence. Recall if you will how easy it is to instill the first medication in a newborn's eyes. But in the seconds it takes to get to the other eye he has already learned that the medication is unpleasant and has it squinched up as tight as wax.

He learns just as quickly to eat solid food. I tell my patients to mix the cereal very thin at first and to use an after dinner coffee spoon instead of their own baby spoon or one that relatives provide to the newly arrived one. When the spoon touches baby's lips he will suck the contents out and before you know it he will be eating.

Fourth, I should like to mention the matter of sensitivity. In my series I have encountered some cases of sensitivity. I am inclined to agree with Dr. Sackett that we shall have less of it when we offer a more varied menu. I am not qualified to discuss the subject even if I thought it posed any great problem. I should like to describe some interesting cases for you to tell you of my very personal series of one who is one year old now. However, suffice it to say that I feel Dr. Sackett has worked out a system of infant care that is safe, simple and satisfactory. I hope more of you will give it a trial and report your results so that next time he can report 3,000 cases instead of this rather imposing 300 personal cases.

Dr. John E. Crews, Orlando, Fla.—About two years ago I began feeding my babies cereal, banana, fruits, and vegetables at six weeks of age. This program was so successful that one year ago I instituted a three-meal-a-day program, beginning when the infants were three weeks old. At first there was an extreme amount of criticism from some of the parents, from most of the grandparents, and in general from most other physicians. Most of the criticism from the parents and the grandparents has been largely overcome. However, there are still many physicians who object to this program. The following is a schedule which I give to the mother when the baby is three weeks old:

6:00 a.m. (1) Cereal: cerevim® or pabeena.® Begin with one tablespoon, increase as tolerated. (2) Banana. Be sure it is ripe. Up to one-half banana. Whip with fork until light. (3) As much formula as baby desires.

9:00 a.m. Orange juice. Strain and dilute with equal amounts of water, gradually increase to full strength. Give baby up to two ounces. Multi-vitamin drops.

12:00 noon. (1) One or two vegetables: choice of spinach, carrots, peas, string beans. Warm and season very lightly with butter or oleomargarine and salt. Baby may have as much as one-half jar of each. (2) As much formula as baby desires.

3:00 p.m. Orange juice, same as 9:00 a.m.

6:00 p.m. (1) One or two fruits: choice of pears, peaches, apricots, prunes, applesauce. Give as much as baby desires. (2) Cereal as for breakfast. (3) As much formula as baby desires.

Offer your baby water three or four times daily between meals. After the evening meal, do not give the baby formula or water until breakfast the next morning. Keep the baby's diapers dry during the night and he will sleep better. If you will follow the program persistently for one week your baby will be sleeping through the entire night. Remember the success of this program depends upon the parents, not upon the baby.

Mothers who wish to nurse their babies can very easily do so. Their breasts can very easily be trained to this three-meal-a-day program. At first during the night it was difficult for some mothers as their breasts did become a little engorged and uncomfortable. However, if the mother would relieve the engorgement by pumping the breast until the tension was relieved, the breast would soon be trained satisfactorily to the three nursings a day and none during the night. In general the babies on three meals a day have fewer feeding problems, have less colic, and appear to be happier babies.

It is my contention that babies, even though they are fed on a three or four-hour schedule of nothing but milk for several months are still hungry babies, even though their weight gain may be satisfactory. In my series of about 100 babies on the three-meal-a-day program, all have gained weight satisfactorily, are happier babies, and they seem to be less susceptible to upper respiratory infections. A baby in this series, at three and one-half weeks of age had an operation for pyloric stenosis. Ten days later he was placed on the above three-meal-a-day program and has progressed satisfactorily in weight and growth. Another baby in this series was started on the three-meal-a-day program at seven days. This baby nursed the breast at midnight for one week. Since then the baby has been on the strict three-meal-a-day program.

I wish to commend Dr. Sackett for his program of early feeding. Its results are very satisfactory.

Dr. Sackett (closing).—With my dietary schedule, I encounter no more allergies than I have seen before in babies on other feeding routines.

Dr. Cole said something about the consistency of food. It has been my observation on one of my own babies and in my practice that these babies handle thicker foods better than foods of a watery consistency. Routinely I advise my mothers to make the foods, particularly the cereals, of a consistency to their liking and then rapidly to thicken it. Most of them will report back, as in my own personal experience, that newborns can take cereal from the spoon in a consistency similar to putty.

Concerning the giving of orange juice in midmorning and midafternoon, I could give Dr. Crews a sly dig. If you once start them on orange juice in the middle of the morning the next thing you know when they are a year or two or three old, there will be adding a cookie, or a slice of bread to the orange juice. This practice could result in a schedule with a three-hour interval.

THE DEVELOPMENT OF VISION AND ITS RELATION TO MUSCLE ANOMALIES*

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The treatment of strabismus in children is based on a combined knowledge of (1) the

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development of normal binocular vision, (2) the effect of visual development on strabismus, and (3) the effect of strabismus on the development of vision. The purpose of this communication is to assemble certain of these facts, the interrelationships of which are not always clear, and to integrate them in such a manner as to provide a sounder background for the rational treatment of strabismus.

NORMAL DEVELOPMENT OF VISION

At birth, ocular function is limited to aimless movements of the eyes. Over the next several years *(Table 1)* the eyes grow as the child grows and vision becomes a fully developed function. This cannot occur, however, until fixation, acuity and fusion have become perfected through learning and practice, since the normal development of vision depends upon their perfection and interrelationship *(Fig. 1)*. It is usually completed by the end of the sixth year of life.

Development of Fixation.—When the eyes of a 2-week-old infant try to fixate an object, they display only incoordinate disjunctive movements. Light stimuli passing through the pupils cause sensory impressions which are conveyed to the ocular motor nuclei by reflex pathways. The resulting efferent impulses to the extraocular muscles cause the eyes to move toward the source of light. At first these reflex fixation movements are inexact and the eyes simply swing pendulum-like until light strikes the foveal areas, which at this age are still undeveloped.

By the end of the sixth week of life an infant, when he stares at a massive object, is able to hold fixation for a few seconds. By a process of continuing adjustment for foveal fixation an exact binocular fixation ability usually develops between the third and fifth months. By this time the foveal areas have developed physiologically as well as anatomically. The eyes fixate simultaneously and follow objects, and the child can reach for them fairly accurately, although his visual acuity is only about 20/2000.

Development of Visual Acuity.—If reflex fixation movements develop and become firmly established, the two eyes will remain parallel most of the time. Once this happens, vision begins to develop. Good fixation, however, is essential for the development of visual acuity. About the sixth month of life the fields of fixation parallel those of an adult except for extreme versions. Gesell and his coworkers[1] studied the ability of infants to fixate by dangling rings from strings in front of them and checking the length of time it took the eyes to move about in an effort to fixate them. When fixation was accomplished, the interval for which it was held was timed. As the children grew older, it was found that they were able to fixate more quickly and hold fixation for longer periods.

Visual acuity in infants has been estimated by noting their response to objects of various sizes placed within their range of vision.[2] At 6 months it is about 20/1000. It is 20/200 at

STEPS IN DEVELOPMENT OF VISION*

Age	Visual Acuity	Fixation and Stability
Birth		Aimless movements
2 weeks		Momentary fixation
6 weeks		Fixation held for a few seconds
5 months	20/2000	Exact fixation held
6 months	20/1000	Normal field of fixation
9 months	20/200	
2 years	20/45	Fusion reflex recognized; capable of complete extinction
3 years	20/30	Fusion reflex established; deteriorates greatly from disuse
4 years	20/20	Fusion reflex and visual acuity deteriorate from disuse; rapid recovery with re-use
6 years		Fusion reflex and visual acuity more stable, may still deteriorate from disuse
8 years		Fusion reflex and visual acuity unconditionally fixed

*Modified from Lyle.[2]

TABLE 1

FACTORS IN THE NORMAL DEVELOPMENT OF VISION

Fixation ⇌ Visual Acuity ⟶ Fusion

FIG. 1

Essential processes in the normal development of vision.

9 months and 20/150 at 12 months. By this time the fixation reflex has become very active through usage and the acquisition of progressively greater skill, and as time passes it becomes still more accurate and refined. At the age of 2 years visual acuity is 20/45. Under the age of 3 years testing can be done with toys of various sizes, placed about 20 feet away. Response to an object with a diameter of ¾ inch is considered equivalent to a visual acuity of approximately 20/80 and response to an object with a diameter of 1.5 inches is considered equivalent to a visual acuity of approximately 20/200. In older children the illiterate "E" or Snellen test types are satisfactory for testing visual acuity.

While fixation improves acuity, acuity also improves the quality of fixation. The development of better fixation and the development of better acuity take place at the same time and in each eye.

Development of Fusion.—Since the fixation reflex is monocular, an image need fall on the retina of only one eye for the adjustment of fixation to be made. The innervation required for moving the fixing eye to the position of fixation, however, always goes to both eyes, which then act as a single organ. It is not possible to fix with one eye without a response by the other eye. This holds even if one eye is covered: the innervation which moves the fixing eye into position when the two eyes are ideally coordinated moves the other eye into the same position of fixation. Actually, coordination is never perfectly precise. There is always some deviation by the non-fixing eye, though if it is slight, it is disregarded and the eyes are said to be orthophoric.

The slight deviation always present in the non-fixing eye, requires another reflex for its correction if both eyes are to see an object at the same time. Whenever the images seen by the two eyes are not on corresponding points of the retinas, a reflex adjustment is made to correct the position of the eyes, so that the images fall on the proper retinal areas and binocular fixation is possible. This is the fusion reflex, whose function it is to correct any imperfections in the coordination of the two eyes.

The fusion reflex is first recognized at 2 years of age, at which time it is capable of complete extinction. Because each eye must look at the same point, it is essential that there be a constant and exact coordination of ocular movements. A very elaborate neuromuscular mechanism exists for this purpose, of which the neurologic component is the more important, since it controls not only the general movements of the eyes but also the minute coordination of these movements. This adjustment mechanism also has the capacity to overcome many anatomic obstacles of considerable degree.

By the age of 3 years the fusion reflexes are becoming established, though they still suffer readily and seriously from disuse. Visual acuity is now 20/30. At the age of 4 years it is 20/20 and extreme versions can be sustained. The fusion reflexes are grounded, and while disuse may cause deterioration, it cannot cause extinction. Any loss sustained after this age can be rapidly overcome by re-use. The adjusting mechanism of the fusional reflex is capable of increasing its power of coordination and regulation with practice. Greater skill is thus acquired.

The amount of imperfection in the coordination of the two eyes which can be corrected by the fusion reflex is termed the amplitude of fusion.[3] It is measured in prism diopter units *(Table 2)*. It varies from person to person, depending, since it is a functional development, upon the skill acquired through usage and practice. When the fusion reflex corrects the deviation of the two eyes, the deviation remains latent and the condition is called heterophoria *(Fig. 2)*. When the amplitude of fusion is not great enough to correct the deviation, the deviation is manifest and the condition is called heterotropia or strabismus *(Fig. 3)*.

By the age of 5 years the fusion reflexes

NORMAL AMPLITUDE OF FUSION*

6 meters	18-20 prism diopters of convergence 6-8 prism diopters of divergence 1-3 prism diopters of vertical vergence 1-2° of cyclovergence
33 cm.†	36-40 prism diopters of convergence 13 prism diopters of divergence

*According to Burian.[3]

†18 prism diopters of convergence are required to change binocular fixation from 6 meters to 33 cm. when the interpupillary distance is 60 mm.

TABLE 2

have assumed unconditional fixity, though they are still capable of some deterioration from disuse. After the eighth year they become unconditionally fixed and the period of instability has ended.

Thus during the first few years of life the child learns to see. His reflexes become perfected and thereafter the normal development of vision is dependent upon the continued use of both eyes.

Physiologic Deviations.—Up to the age of 6 to 8 months, during the development of ocular movements, it is perfectly normal for the eyes to deviate from time to time. Some abnormal muscular movements, in fact, continue up to the age of 18 months. They are only transient, however, and they have certain characteristics[4] which clearly differentiate them, at least in most instances, from true strabismus: they last only a few seconds. They are usually binocular. The direction of the deviation is variable. They occur because the

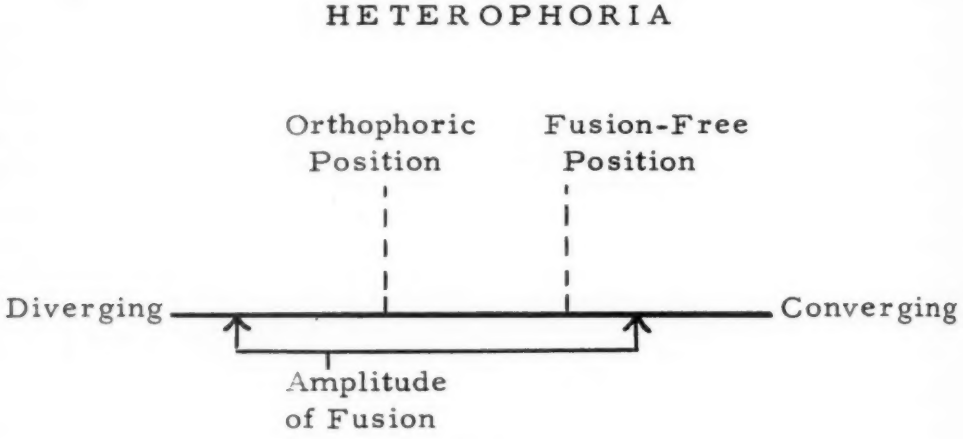

FIG. 2

Diagrammatic showing of heterophoria. Amplitude of fusion includes both the orthophoric and the fusion-free position of the eyes. The deviation remains latent (convergent or divergent).

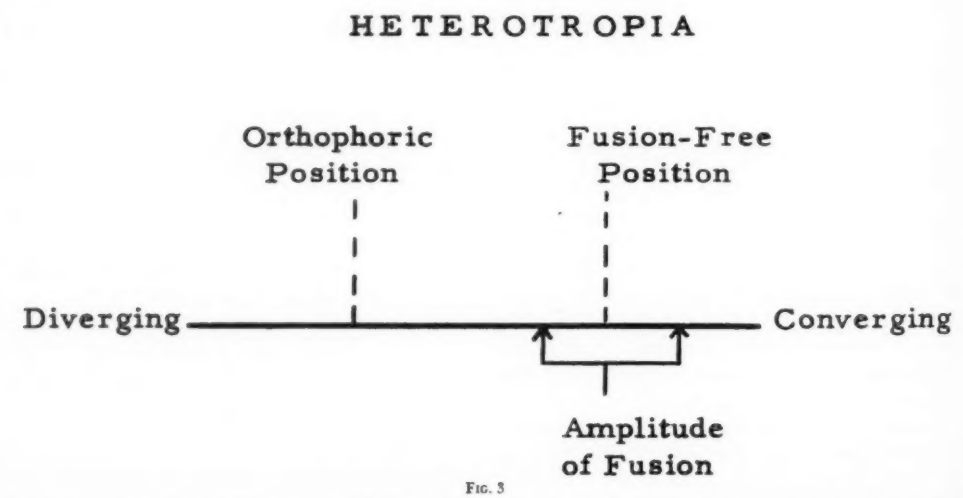

FIG. 3

Diagrammatic showing of heterotropia. Amplitude of fusion includes only the fusion-free position of the eyes. The amplitude is not great enough to include the orthophoric position also. The deviation is manifest (strabismus, convergent or divergent).

neuromuscular ocular mechanism for maintaining binocular vision is still undeveloped and there is temporary insufficiency of the coordination centers. These deviations do not result in strabismus. On the contrary, the children who present them usually develop normal binocular vision. On the other hand, any constant deviation in one direction which persists over 30 seconds in a child over 18 months of age may be considered a true strabismus.

EFFECT OF OBSTACLES ON NORMAL DEVELOPMENT OF VISION

In the absence of abnormal conditions, the visual apparatus develops normally and the child learns to use his eyes correctly. This does not always happen, however, because obstacles may arise which make correct usage difficult or impossible. When this happens, the normal development of vision is prevented. If some obstacle prevents the use of an eye, it will not develop a good, accurate fixation reflex. Vision will be poor and the fusion reflex will not develop. If the difficulty arises after birth, the development of vision will be arrested. If the obstruction is allowed to continue, the arrest of visual development may be followed by a deterioration of whatever vision has already been acquired. If the obstacle is monocular, the result of poor vision is strabismus. If it is binocular, nystagmus may result. If the obstruction has been corrected, the development of vision which has been arrested will be resumed and will continue up to about the age of 6 years. Up to that time fixation, visual acuity and fusion are labile and can be influenced, though the final amount of development is dependent upon the duration of the obstruction prior to its correction.

Obstacles to the normal development of vision may occur in any part of the visual apparatus, as follows:

(1) Optical: high refractive errors (myopia, hyperopia, astigmatism); incorrect glasses; anisometropia; opacites of the cornea, lens or vitreous.

(2) Sensory: failure of development of visual pathways; tumors or diseases of the retina or optic nerve; prolonged covering of one eye with an eye patch; reflex closure of the lids as the result of inflammation; congenital ptosis; congenital anomalies of the retina or disc.

(3) Motor: conditions affecting the adnexa, orbit and globe (inflammatory or neoplastic); conditions affecting the muscle itself (birth trauma or injuries); fibrosis of the muscle; anomalous insertions of the extraocular muscles; conditions affecting the motor nerve trunk, the motor nerve nuclei and the supranuclear centers either singly or in combination.

(4) Central: physical or mental trauma or illness in a child with poor fusion sense; deficiency of fusion.

EFFECT OF VISION ON STRABISMUS AND THE POSITION OF THE EYES

All varieties of strabismus are produced by some obstacle which exists prior to the development of normal binocular vision. Children with organic ocular defects which either are not corrected or which cannot be corrected always present faulty development of fixation and visual acuity, and these deficiencies prevent, in turn, development of the fusion reflex. Faulty binocular vision may cause strabismus.

The position of the squinting eye usually depends upon the age at which the obstruction to vision occurs. An eye with poor vision in infancy and childhood will usually turn inward. If poor vision comes on in adolescence, the eye will often be straight. If it comes on in adult life, the eye will usually turn out. As a rule, the older the person, the greater is the tendency to outward deviations. There are, however, numerous exceptions to these generalizations.

EFFECT OF REFRACTIVE OBSTACLES ON VISION

Of all the obstacles to normal vision in a child, hypermetropia is the most obvious and the best understood. When a person with normal eyes looks at a distant object, the eyes are parallel and accommodation is not necessary. When he looks at a near object, the eyes turn inward and he accommodates so that he can see more clearly. There is always a definite convergence-accommodation ratio. When, however, the child who is far-sighted looks into the distance, he must accommodate in order to see clearly. When he looks at a near object, he must accommodate still more in

order to see clearly. Thus he must accommodate both for distant and for near objects in his endeavor to obtain a clear image. The need for extra accommodation causes abnormal convergence and the eyes move inward.

The conditions in myopia are exactly opposite. When the child looks at a near object, he need make only a small amount of accommodative effort in order to see it clearly. Without such an effort, there is failure of convergence and the eyes drift outward. Some myopic infants, however, do not discover that accommodation makes vision more blurred. With increased attempts at accommodation, there is increased convergence and the eyes move inward.

If the fusional reflex is well developed, it is possible, in both hypermetropia and myopia, for the child to overcome the tendency to misalignment of the eyes either partly or completely while he sees images clearly. If this happens, the eyes will remain properly aligned for binocular vision. If, however, the fusional reflex is poorly developed, one eye may begin to deviate when he is tired or ill. Deviation is often precipitated by some one of the childhood diseases, or by an emotional strain. Whatever the cause, it may become progressively worse. Moreover, the deviation causes double vision and visual confusion. In his endeavor to overcome these difficulties, the child presents the complications of strabismus. He suppresses with the deviated eye, and, if he does not alternate fixation, the visual acuity of the deviating eye will begin to deteriorate from disuse. The end result may be amblyopia ex anopsia or even abnormal retinal correspondence.

ANATOMIC CHANGES

Every child with strabismus resulting from a refractive obstacle which has been neglected will ultimately develop anatomic changes. These changes will occur even though the eyes may originally have had normal extraocular muscles, fascia, and functioning neuromuscular apparatus. They are the result of the constant contraction and spasm of the overstimulated muscle on the side of the deviation. The spastic muscle becomes hypertrophied and eventually some degree of fibrosis develops. The antagonistic muscle, because it is in a constant state of relaxation, becomes thinned out and permanently weakened. These anatomic changes are irreversible. Once they have occurred, surgery is necessary to replace the eye in proper alignment.

EFFECT OF STRABISMUS ON VISION

Strabismus exerts a number of consecutive deleterious effects on vision. Visual confusion and diplopia are followed by suppression in one eye. Suppression may be followed by amblyopia ex anopsia. Amblyopia may be followed by abnormal retinal correspondence, which may be followed by eccentric fixation *(Fig. 4)*. The whole chain of consequences is not evident in the majority of cases, but when all of these complications occur, the order of their development is invariably the same. Each successive complication is an adjustment to the preceding complication. The extent of the effect of strabismus on vision is thus dependent on the duration of the strabismus.

Suppression.—Strabismus which occurs in the absence of organic ocular defects is usual-

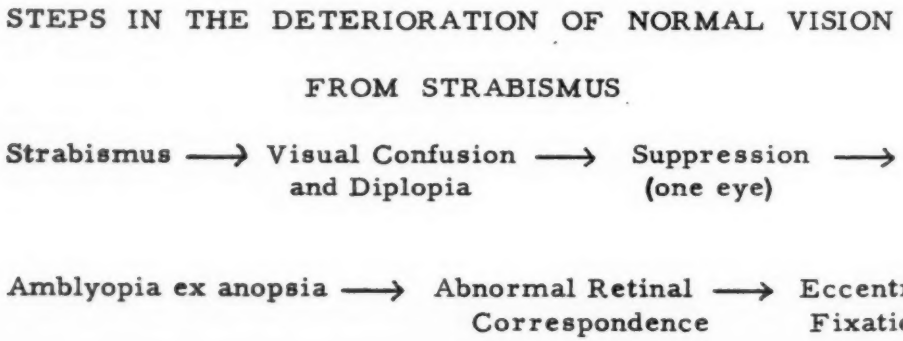

FIG. 4

Progressive steps in the deterioration of normal vision as the result of strabismus.

ly caused by a deficiency of fusion. It begins, as a rule, at about 2 years of age, when the development of the fusion reflex becomes evident and when the visual acuity is around 20/40. When there is failure of fusion, one eye drifts in one direction and the other in another, with the result that they fixate different objects, and there is visual confusion and diplopia. The child may adjust to this abnormal condition by suppressing the image received by one eye. There are usually, therefore, two scotomas of suppression in the non-fixing eye *(Fig. 5)*. The first, the macular area of the non-fixing eye, eliminates visual confusion. The second, which corresponds to the visual direction of the fovea of the fixing eye, eliminates diplopia. In some cases of esotropia, the object seen by the fovea of the fixing eye may fall on the optic disc of the deviating eye, and cause only one suppression scotoma.

Amblyopia ex Anopsia.—Because of continued suppression and disuse of the squinting eye, visual acuity begins to deteriorate. This development is the forerunner of amblyopia of disuse (amblyopia ex anopsia or suppression amblyopia).[5] The longer the duration of the squint, the more pronounced is the suppression and the more difficult is the correction of the amblyopia.

About a third of all patients with strabismus present amblyopia ex anopsia.[6] Its incidence varies with circumstances. It is less frequent in alternating than in monocular strabismus because each fovea is used alternately. It is less frequent in periodic strabismus, or does not occur at all, because there is opportunity for binocular vision. It is more frequent in constant strabismus. It is less likely to be present in a child who presents visual confusion because in such cases suppression is incomplete. Amblyopia is also less frequent in exotropia, in which visual confusion is more frequent, than in esotropia, in which suppression is more frequent. The incidence of amblyopia, finally, increases directly with the degree of refractive error and anisometropia.

Visual acuity can greatly alter the function of the extraocular muscles. Amblyopic eyes have poor powers of abduction. As acuity improves, abduction becomes more normal. When an amblyopic eye fixates, the deviation is greater than when the normal eye fixates. Children who present strabismus with amblyopia therefore are likely to present both primary and secondary deviations. Even if they are not treated, some children seem to lose their strabismus as they grow older, though amblyopia continues to be present in such cases and there will be poor vision in one eye.

Abnormal Retinal Correspondence.—Some

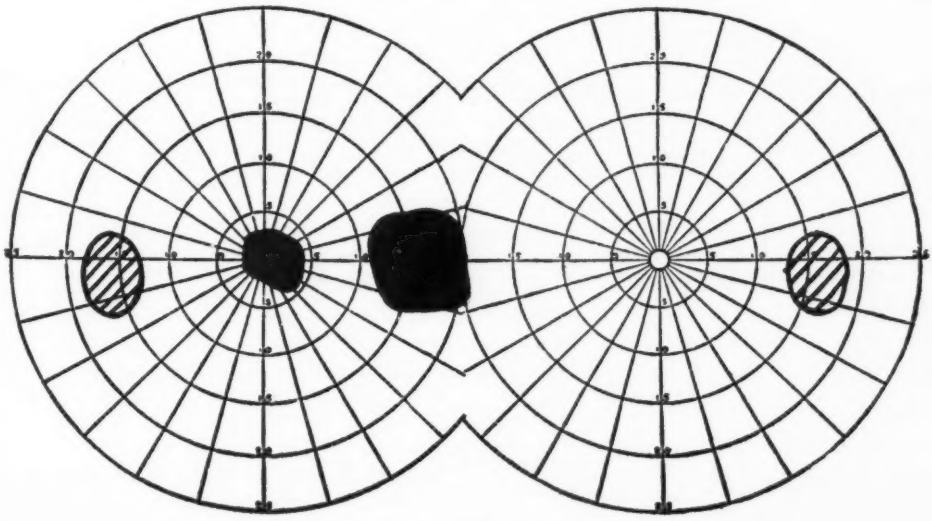

FIG. 5

Scotomas of squinting eye as plotted by binocular, red-green filter projection. The right eye is the fixing eye.

eyes which have developed suppression and amblyopia nevertheless attempt to adjust further to the position of strabismus. The squinting eye develops a functional area (false fovea) in the retina which assumes a common visual direction with the fovea of the fixing eye. Its development is an attempt on the part of the squinting eye to function binocularly with the fixing eye. The visual direction of the fovea of the squinting eye is changed, and the two foveal areas no longer have a common visual direction. Normal retinal correspondence of the two eyes is lost and is replaced by abnormal retinal correspondence. The visual acuity of the true fovea of the squinting eye, however, is better than that of the false fovea, and if the squinting eye is forced to fixate, it will do so with the true fovea (central fixation).

Eccentric Fixation.—When there is organic involvement of the fovea of the squinting eye, or when the amblyopia of the suppression has been of long duration, the visual acuity of the fovea may deteriorate to a level lower than that of the false fovea. Then the extrafoveal area of the retina will have better vision than the fovea itself. Under these circumstances the squinting eye will fixate with its false fovea in monocular fixation (eccentric fixation).

PRINCIPLES OF TREATMENT

In order that the child may develop stable habits of binocular vision he must have good acuity in each eye, good motility of each eye, and proper alignment of the two eyes during early childhood. Treatment should be based on the following principles:

(1) Refractive obstacles should be identified and corrected as soon as strabismus be-

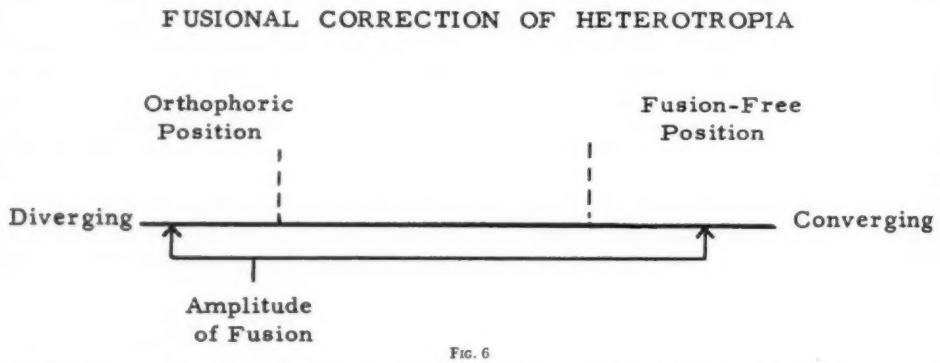

FIG. 6

Fusion correction of heterotropia. The increased fusional amplitude includes both the orthophoric and the fusion-free position of the eyes. The deviation remains latent (convergent or divergent).

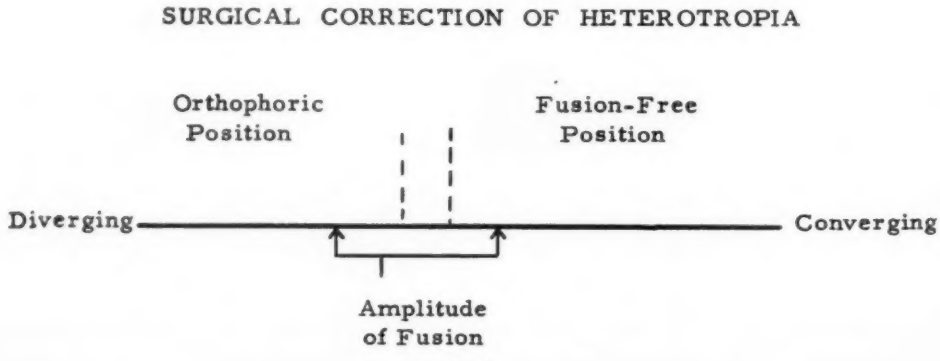

FIG. 7

Surgical correction of heterotropia. Surgery has brought the fusion-free position to a more orthophoric position, allowing the amplitude of fusion to include both. The deviation remains latent (convergent or divergent).

comes evident. This plan of procedure may also correct the deviation, prevent deterioration of vision, and provide opportunities for further and more skillful development of fusion amplitudes.

(2) If strabismus has been present for a period of time and vision has deteriorated, the complications of the squint will require treatment, in addition to treatment of the refractive obstacle. If eccentric fixation or abnormal retinal correspondence is present, the fixing eye should be totally occluded and the fovea of the squinting eye should be stimulated in an effort to re-establish normal retinal correspondence.

(3) Amblyopia ex anopsia will also require treatment. Therapy includes forcing the use of the amblyopic eye, creating interest in its use, and encouraging the patient to undertake activities which require its use. It should be stimulated. Total occlusion of the fixing eye is imperative until visual acuity has improved to 20/70. Partial occlusion is then employed until acuity is 20/20, after which part-time occlusion is used only to make visual acuity more stable. The best possible vision should be maintained until fusion and alignment have been gained.

(4) Treatment of suppression of the squinting eye is directed toward removal of the scotomas and the re-establishment of awareness of the diplopia.

(5) Treatment of the deficiency in amplitude of fusion is necesary, so that the fusion reflex may gain greater power and skill. The amplitude should be increased so that it will include the fusion-free position of the eyes and the orthophoric position, with ample reserve for comfort *(Fig. 6)*. Surgery on the extraocular muscles may be necessary in order to place the eyes in proper alignment. This requires moving the fusion-free position to a more orthophoric position. Then the amplitude of fusion may be increased to include both. The deviation now becomes latent rather than manifest *(Fig. 7)*.

(6) If amblyopia ex anopsia is superimposed, because of disuse, on an organic defect, its treatment should still be attempted. All organic ocular defects should be corrected as promptly as possible, so that the child may use his eyes and develop stable binocular vision by learning and practice.

(7) Regardless of what condition or conditions may be present, treatment should always be begun as early as possible, for the reason that the development of acuity and fusion is labile only for the first 6 years of life. Another reason for prompt treatment is that the longer amblyopia has been present, the more difficult it is to treat. If treatment is delayed, the child will acquire abnormal ways of seeing, and the possibility of acquiring normal ways may be lost. If treatment is deferred until after 6 years of age, the results will probably be limited to cosmetic improvement. This is, of course, a considerable gain, but it falls far short of the ideal of normal binocular vision.

SUMMARY

The development of vision is a continuous process, which involves learning and practice. The development of fixation and of acuity proceed simultaneously. The development of fusion ultimately gives stable binocular vision. The most rapid development of vision occurs during the first 2 years of life. An acuity of 20/2000 at 5 months improves to about 20/45 at 2 years. A fusion reflex is evident at 2 years. It may become capable of correcting existing deviations and overcoming certain anatomic obstacles. The development of normal binocular vision (20/20 visual acuity with fusion) is usually complete by the end of the sixth year.

When obstacles arise, as they frequently do, which make it difficult or impossible to use the visual apparatus correctly, there is faulty development of vision, which may result in strabismus. More often, strabismus is the result of faulty development of the fusion reflex. Frequently it is the cause of visual deterioration. Suppression may lead to amblyopia ex anopsia. Abnormal retinal correspondence and eccentric fixation may result.

The treatment of strabismus includes the development or restoration of central fixation and visual acuity with provision for the fusion reflex to develop amplitude for stable binocular vision. The ocular misalignment caused by irreversible muscle changes which result from uncorrected deviations, can be eliminated only by surgery. Since the principles of treatment are based on the basic physiology of binocular vision, it is essential that

treatment of strabismus be begun before the potentials of such vision are lost.

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THE DIFFERENTIAL DIAGNOSIS OF DEAFNESS*

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The differential diagnosis of deafness, which implies the identification of the etiologic factor or factors responsible for it, requires two preliminary steps. The first is the establishment of the existence of the hearing defect and of its approximate degree, by history, physical examination, local examination, and investigation of the auditory function. The second is the classification of the defect as conductive, perceptive, or mixed.

GENERAL INVESTIGATION

The differential diagnosis of deafness begins with exactly the same careful history which would be taken in any other general examination. Particular attention is paid to the possible existence of systemic disease, the use of drugs, and mental and emotional traits and habits.

The auditory history is similarly complete. Special attention is paid to familial hearing defects and the possible relation of the hearing disability to previous disease, physiologic function, environment, and other factors. All details of the hearing loss are inquired into: whether it is unilateral or bilateral, and, if it is bilateral, whether it is worse on one side than the other; how long it has lasted; whether it has progressed; whether it is intermittent or constant; whether the loss is general or is limited to particular sounds or to speech; the precise circumstances of the onset; whether the defect is related to other symptoms or signs, particularly pain in or about the ears, headache, earache and an aural discharge; whether it is associated with tinnitus. If any of these abnormalities exist, they are themselves investigated in detail.

The otologist who secures a history of this sort, in the detail indicated, will often find that he already has in his hands the clue to both the diagnosis and the differential diagnosis of the hearing defect which he is investigating.

The otologist is naturally not trained or equipped to make a detailed physical investigation, but he can make a sufficiently complete examination to detect gross abnormalities and to recognize the need for consultation with an internist or other specialist.

After the usual inspection, palpation, and manipulation of the external ear and mastoid region, the status of the external auditory canal and ear drum is determined with the aid of the head mirror and speculum, supplemented by the electric otoscope. The mobility of the drum and ossicles is tested by means of Siegel's pneumatic speculum. The eustachian tube is inflated to determine its patency, the presence of abnormal secretions in the tympanum, and the existence of possible perforations.

The local examination is concluded with a similarly detailed investigation of the nose and throat. Special attention is paid to the nasal, nasopharyngeal and paranasal spaces, since these areas are often the indirect source of the hearing defect.

The general examination is concluded with whatever laboratory tests may be indicated. Cytologic studies should always be carried out.

INVESTIGATION OF THE AUDITORY FUNCTION

Whispered and spoken voice tests are performed merely as a matter of routine. Neither type of test is accurate and neither is of differential diagnostic value. Tuning fork tests and audiometry provide the real information necessary for diagnosis.

Tuning fork tests provide the most generally useful method of determining whether

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a hearing defect is present and whether it is conductive, perceptive or mixed. The forks are cheap, they are usable anywhere and at any time, and they require no upkeep. With them it is possible to determine relative auditory acuity for tones of high and low pitch, as well as the duration of air and bone conduction. It is unfortunate that their usefulness is not more widely recognized.

Tests with forks whose double vibrations vary from 16 to 4096 per second are carried out for the upper and lower limits of hearing for tones. In addition, the following special tests are employed:

(1) The Weber test, which determines, by comparison of the bone conduction on each side, whether monaural deafness is of nerve or conductive origin. This test is useful only to corroborate other findings.

(2) The Schwabach test, which compares the duration of the patient's bone conduction with that of the examiner, whose hearing is presumably normal. Masking is used in this test.

(3) The Rinne test, which compares the duration of bone conduction and air conduction in the ear under investigation. This is a relatively informative test. It is considered negative (and the hearing loss is assumed to be of the conductive type) when air conduction is shortened. It is considered positive (and the hearing loss is assumed to be of the perceptive type) when bone conduction is shortened. When the duration of bone and of air conduction are approximately equal, it will usually be found that while both are shortened, air conduction has sustained the greater loss. In such cases either a perceptive or a mixed type of deafness is present.

Because the audiometer is a precision instrument, it produces tones of greater intensity and of longer duration than can be produced by tuning forks. It therefore permits rapid testing by both air and bone conduction through the usable range of tones, with measurement of any loss in units of sensation. One of its greatest advantages is that the record of testing is permanent, which permits successive audiograms to be compared with each other.

The curve of the audiometric graph usually indicates whether the hearing loss is conductive, perceptive or mixed. In pure conductive deafness the low tone loss is ordinarily the greater and bone conduction is normal for the frequencies 512, 1024 and 2048 cycles. In pure nerve deafness there is ordinarily relatively good hearing by air conduction for the low tones, and progressively poorer hearing for the higher frequencies. Bone conduction loss parallels air conduction loss. In mixed deafness there is loss of hearing by air conduction, usually paralleled by a similar loss by bone conduction, for both low and high tones; the audiometric curve begins well below the zero line in the low tones and drops progressively toward the high tones.

CLASSIFICATION OF DEAFNESS

Generally speaking, the three varieties of deafness are characterized by definite and clear cut findings and arise on different etiologic backgrounds:

In conductive deafness: (1) Hearing by air conduction is better for high than for low tones. (2) The lower tone limit may be raised, so that tones of very low frequency may not be heard at all. (3) In advanced cases loss for both high and low tones is approximately equal and the audiometric curve is flat. (4) Bone conduction is normal. (5) The Rinne test is negative. Possible causes include chronic obstruction of the eustachian tube from any one of a number of causes, acute or chronic suppurative otitis media, secretory otitis media, congenital atresia of the auditory canal, perforations of the tympanum, and otosclerosis.

In perceptive deafness: (1) hearing by air conduction is relatively better for low than for high tones. (2) The upper tone limit may be lowered, so that very high tones are not heard at all. (3) Bone conduction loss parallels air conduction loss. (4) The Rinne test is positive, the duration of air conduction with tuning forks being longer, and that of bone conduction being shorter, than in the normal subject. Possible causes include auditory neuritis following partial or complete destruction of the labyrinth from various causes, traumatic destruction of the internal ear or auditory nerve, syphilis, acoustic trauma, psychosomatic causes, acoustic neuroma, Menière's disease, and deaf mutism.

Mixed deafness, which may occur in a variety of conditions but is most common in long standing middle ear disease, presents

the characteristics of both conductive and perceptive deafness: (1) There is marked loss by air conduction for all frequencies, with added and increasing loss in the high frequencies. (2) Tones of low frequency are heard fairly well by bone conduction, but hearing for higher frequencies is either lost or is greatly impaired. (3) The Rinne test is likely to be negative when fork 256 or 512 d.v. is used. (4) The Weber test with fork 256 d.v. shows lateralization to the affected (or more severely affected) side. (5) When neural degeneration is superimposed on a conductive lesion, the highest tones are affected to a much greater degree than the low tones, and for all practical purposes the higher components of speech tones are almost entirely lost.

DIFFERENTIAL DIAGNOSTIC CONSIDERATIONS

When the data obtained from the history, the general and local physical examinations, and the test of auditory function have been evaluated and have been supplemented by such laboratory tests as are indicated in the special case, it is usually possible for the otologist to determine, with a fair degree of certainty, the type of deafness present, as well as, in many cases, to identify the etiologic factor responsible for it. It should be emphasized, however, that the differential diagnosis of the hearing defect depends upon an evaluation of all the available data and not upon the results of any single test.

The types of deafness most often encountered are caused, in the order of frequency, by infections of the middle ear and its appendages, presbycusis, partial or intermittent obstruction of the eustachian tubes by lymphoid tissue in the nasopharynx (high tone deafness), and otosclerosis. Deafness of other origins, however, while it is considerably less frequent, cannot be ignored in the differential diagnosis of any auditory impairment which cannot promptly be assigned to any of the causes just listed.

Middle Ear Infections.—Middle ear deafness, which is usually unilateral, is ordinarily easy to diagnose because the history makes its origin apparent. The physical findings are also more clear cut than they are in deafness of most other origins. There are three possible clinical courses: (1) The auditory impairment may occur promptly after an acute suppurative infection. (2) It may appear later, after chronic inflammatory changes have been superimposed on the acute infection. (3) It may follow repeated subacute infections by micro-organisms of low virulence. The degree of auditory impairment is related to the severity of the pathologic process. In severe acute disease there may be actual destruction of portions of the auditory apparatus, with later formation of fibrous tissue. In chronic cases the most important pathologic change is narrowing of the eustachian tube, with stricture formation. Coincident sinus infections and the development of exuberant lymphoid tissue increase the hearing defect.

Otoscopic examination shows thickening and retraction of the drum. When Siegel's otoscope is used, it is possible to recognize fixation of portions of the tympanic membrane by adhesions. In advanced cases, the presence and location of organized strictures can be recognized by the use of eustachian bougies.

In acute cases the audiometric curve shows a loss of from 30 to 50 decibels for lower tones. The curve is flat. In chronic cases the curve shows a downward slope for high frequencies.

Presbycusis.—Presbycusis is a physiologic, insidious, slowly progressive loss of hearing for high-pitched tones which comes on with advancing age at the rate, it is said, of 1 decibel for each year of life. When it is associated with arteriosclerosis it is likely to be unilateral or to be more pronounced on one side than on the other. Lack of attention and slowness of cerebration in older subjects increase the apparent impairment, but at that, the complaint of deafness is often greater than accurate hearing tests would indicate. The phonetically balanced word test should produce a maximum score of 90 per cent, but in patients with presbycusis it never does. The recruitment phenomenon is consistently present.

Hyperplasia of Nasopharyngeal Lymphoid Tissue.—It is only within recent years that an insidious loss of acuity for high tones (above 8000 d.v.) has been recognized as caused by partial or intermittent obstruction of the eustachian tubes by lymphoid tissue in the nasopharynx. This type of deafness most frequently occurs in children, particularly in children who have a recurrence of lymphoid tissue after removal of the adenoids.

Otosclerosis.—Otosclerosis, which is the most important cause of progressive conductive deafness in adults, affects about 1 per cent of the white adult population. It is seldom observed in the negro. It is the only really common manifestation of sex-linked inherited deafness. It is transmitted as a recessive sex characteristic, according to the classic Mendelian pattern, and occurs in women more often than in men, in the ratio of 3:2. Though successive generations may be affected, the tendency is to skip one or more generations of a family. Puberty and the menopause uniformly accelerate its progress. The effect of pregnancy and of lactation is less consistent. Both ears are eventually affected in all cases, but progression is more rapid on one side than on the other.

Differential diagnosis begins with the confirmation of the conductive nature of the deafness, and with a comparison of air and bone conduction, by the Rinne test, which is negative, and the Schwabach test, which is prolonged. The Weber test lateralizes to the more severely affected side.

The five other principal causes of conductive deafness are next excluded: (1) Acute suppurative otitis media, by the history. (2) Secretory otitis media, by otoscopy, inspection of the nasopharynx, and inflation of the eustachian tubes. (3) Adhesive otitis media, by otoscopy. (4) Perforation of the tympanic membrane, by otoscopy. (5) Occlusion of the external auditory canal, whether by cerumen, a foreign body, a tumor, or atresia, by otoscopy.

Schwartze's sign is usually positive, but it is not necessary for diagnosis. Additional confirmatory facts are a positive family history and the unimpaired quality of the speech; in the uncomplicated case, because bone conduction remains good, speech remains well modulated and well articulated. Tinnitus is an almost constant accompaniment of otosclerotic deafness in the adult. It is usually low and rumbling, but may be high-pitched. Paracusis willisiana is also frequent.

The audiometric picture is typical in uncomplicated otosclerosis: bone conduction is good, but there is a marked hearing impairment for low frequencies by air conduction. The hearing loss is greater for speech than for pure tones, and greater for words with low frequency components than for those falling within the high frequency range. There is a consistent loss in loudness function for all intensities short of the maximum level, and a decided difference in the loudness function of the two ears when the threshold curves are the same. The low tones are invariably affected and the hearing loss is most often uniform for all frequencies, though shallow curves and dips do occur at the higher side of the audiogram.

Arteriosclerosis.—In deafness caused by arteriosclerosis the hearing loss is for all frequencies, because sclerosis of the internal auditory artery, which is the sole vascular supply of the labyrinth, affects the entire cochlea and diminishes its total performance. Secondary nerve degeneration follows, and a greater loss for all frequencies will be shown audiometrically, though it is important, in evaluating the findings, to take into account the slower cerebration of advancing age. Tinnitus, which is almost uniformly present in the affected, or more severely affected, ear furnishes the clue to the etiologic diagnosis.

Infectious Diseases. — Infectious diseases which most often cause deafness include scrub typhus, influenza, rubella, mumps, and syphilis. Of these, the latter three are the most important.

When the mother contracts rubella in the first trimester of pregnancy, the fetal labyrinthine capsule not infrequently is affected and the type of involvement observed in otosclerosis develops. Deafness, which is usually unilateral, is for some unknown reason a complication of some epidemics of mumps but not of others. The audiometric pattern is usually typical of perceptive deafness, though it may suggest otosclerosis. In any event, it is not specific for mumps. Bone conduction remains good and speech, as in otosclerosis, therefore continues well modulated. In all deafness caused by disease the diagnosis is made chiefly by the history of the preceding illness.

The incidence of acquired deaf mutism in syphilis varies with the type of syphilis and with the strain responsible for the disease in specific localities. The strain of spirochete which causes neurosyphilis is apt to

attack the eighth nerve. Females are more often affected than males.

Diagnosis offers no difficulties if a congenitally deaf child, in addition to absence of labyrinthine function (vestibular and cochlear), also presents notched teeth and interstitial keratitis (Hutchinson's triad), or if blood and spinal fluid serologic tests are positive. In the absence of these distinctive signs, diagnosis may be difficult, since the audiogram is indistinguishable from that of acquired deaf mutism from other causes.

Auditory impairment in congenital syphilis is not always evident in infancy. It is sometimes not manifest until the tenth year, and it may not appear until the twenty-fifth year. Then hearing characteristically deteriorates rapidly, in both ears, with complete loss of both cochlear and vestibular function. There is no set pattern of a response to the Barany test: caloric stimulation may produce a hyperactive response, or there may be no response at all to the rotation tests. Responses to all vestibular tests may change from time to time.

In the acquired type of syphilitic deafness the middle ear, mastoid process or eustachian tube may be affected. Audiometric curves are typical of nerve deafness but are not specific.

A therapeutic test may clarify the syphilitic etiology in doubtful cases, and may perhaps prevent further extension of the auditory impairment, but the pathologic process itself is irreversible.

Psychogenic Deafness.—Psychogenic deafness occurs in the patient who is emotionally unstable, inherently psychoneurotic in his make-up, and unable to adjust himself either to the ordinary situations of life or to situations of special difficulty. Since it is essentially an escape mechanism, it was particularly frequent, as might have been expected, in the Army in World War II. The hearing loss, which is bilateral and complete or almost complete, may come on insidiously, but more often it follows some sudden shock or some unpleasant or dangerous situation.

The confused findings are a clue to the diagnosis. Tuning fork and other tests give inconsistent results. Vestibular responses are normal. Audiograms show bizarre patterns when they are first made and out-of-line patterns when they are repeated. The hearing loss is not consistent with the otologic picture. There is an equal loss for both bone and air conduction. A person who claims to be totally deaf is annoyed by loud noises. The voice remains unchanged. A hearing aid may not give the improvement which hearing tests indicate that it should or, to the contrary, the amplification of sound may be beyond all reasonable expectations.

The difficulty of diagnosis lies in the fact that the otologist must rely, for the most part, on the patient's controlled responses. It is significant, however, that the audiometric and other patterns, which tend to be of the same order in true deafness whenever they are made, differ widely in psychogenic deafness. The response to narcosynthesis is at the same time the best method of confirming the diagnosis and of curing the supposed auditory impairment.

Hereditary Origins.—Hereditary deafness is a type in which many factors remain to be clarified. The condition should be suspected in any young child who fails to acquire articulate speech at the normal age. This story, combined with failure to respond to loud sounds or to exhibit a characteristic reaction to vestibular tests, is usually sufficient to establish the diagnosis. The history is of the greatest importance. Hereditary deafness must be distinguished from the type of deafness acquired as the result of intrauterine or neonatal meningitis or labyrinthitis, congenital syphilis, or rubella, or of the use by the mother during pregnancy of such drugs as quinine, lead, arsenic and mercury.

Head Injuries.—In deafness caused by head injuries the history clearly furnishes the most useful clue to diagnosis. Since pre-existing deafness may be aggravated, pre-injury audiograms, if they exist, are of great clinical value, as well as of medicolegal value.

Certain types of head injury are most likely to produce deafness:

(1) A transverse fracture of the petrous pyramid, with destruction of the labyrinth, which causes total deafness on the injured side.

(2) A longitudinal fracture of the skull, which is apt to injure the middle ear. The resulting deafness is usually conductive but may be perceptive or, less often, mixed. Middle ear lesions often heal following trauma, with improvement in, or disappear-

ance of, the auditory loss. In this type of injury the audiometric pattern varies from case to case, and also varies from time to time in the same case.

(3) Concussion of the brain, in which the cochlear nuclei may be damaged and the clinical picture often suggests medullary injury. An audiogram taken immediately after injury will show a practically uniform hearing loss for all frequencies. A few weeks later the curve shows a downward sloping for both high and low tones, with one or two peaks always remaining for middle frequencies by air conduction. Acuity by bone conduction is good for some frequencies and poor for others.

Acoustic Trauma.—The diagnosis of deafness resulting from acoustic trauma rests chiefly upon the history. This type of hearing defect is most frequently seen in persons who work as boiler-makers (in whom it was first recognized), riveters, miners, and drillers, and in similar noisy occupations. Airplane pilots now constitute a large group of such patients. Traumatic deafness may be caused by a loud explosion, a high explosive shell or mine, or a gun of small caliber if the range is sufficiently close. Generally speaking, an explosion within a range of 20 feet or less causes the most damage. Traumatic deafness is, however, a type in which the individual reaction varies.

The hearing loss, which may or may not be accompanied by tinnitus, also depends upon the total time of exposure, the length of time the single exposure (or repeated exposures) lasts, the loudness of the noise, the frequency of the tones, the environment, and the use or non-use of protective devices. The age of the subject and the presence of preexisting auditory impairment also determine the results of acoustic trauma.

Physical examination may be entirely negative, though in the occasional case the tympanic membrane is ruptured, the ossicles are fractured, the intra-aural muscles are torn, and the osseous labyrinth is fractured. The resulting deafness is natually profound. If, on the other hand, the acoustic trauma reaches the ear via the eustachian tubes, the tympanic membrane may be ruptured outward. The cochlea, for obvious reasons, is spared in this type of injury and the hearing loss may be slight. If trauma is serious, there is notable impairment for speech, especially for whispered speech.

The audiometric pattern is characteristic of cochlear deafness, with a notch at the frequency 4096 for both air and bone conduction. If trauma has been slight, there will be no other abnormalities. If it has been severe, or repeated, the loss will extend below the 4000-cycle level. The higher frequencies are always affected first.

Altered Atmospheric Pressures.—The diagnosis of deafness caused by altered atmospheric pressures is simple, since this type of impairment occurs only in those who work in compressed air, in tunnels, caissons and submarines, or as divers, and who are subjected to too great pressure or to sudden changes of pressure. A second important cause of this type of deafness is the change in the nitrogen concentration of the tissues, which alters as the atmospheric pressure is increased. The degree of impairment is frequently an individual response.

In caisson deafness the tympanum may or may not be ruptured. Vertigo and tinnitus are associated. Bone conduction is markedly diminished. If the neural mechanism has been damaged, the Rinne test is positive.

The fact that deafness does not occur immediately after the patient has been at work in a caisson or in some similar place does not necessarily exclude this type of deafness from diagnostic consideration. It is entirely possible for the effects of an increased concentration of nitrogen in the cochlear nuclei to be considerably delayed. If they are, the resulting deafness is of the perceptive type and the audiometric curve is typical of that obtained in concussion of the brain with delayed auditory impairment.

Menière's Disease.—In Menière's disease, which is an episodic condition, severe deafness is associated with attacks of intense vertigo, nausea and vomiting, severe equilibratory disturbances, headache, prostration, nystagmus, and loud tinnitus. Diplopia and loss of consciousness are occasionally present, and some patients suffer from diplacusis, in which several tones sound of louder pitch in one ear than in the other. Attacks occur at irregular intervals, varying from weeks to months and years. They may last from a few minutes to several days or longer. After

severe attacks it may be days or weeks before there is a return to normal status.

Menière's disease may be dismissed from diagnostic consideration unless there is an absolutely definite history of paroxysmal attacks of true whirling vertigo of sudden onset, associated with tinnitus and with progressive perceptive deafness. In the typical case tinnitus and deafness have been present for varying periods of time before these definite episodes occur. The deafness increases with each attack and improves in the interims, but the hearing loss is progressive and will eventually become total in uninterrupted cases. In half or more of all patients attacks of vertigo cease when total deafness ensues.

The investigation of patients with Menière's disease gives inconsistent results, as might be expected in such a condition. The static labyrinth appears to be functioning normally, or subnormally, or not at all, depending upon the stage of the disease at which the study is made. The audiometric pattern is equally inconsistent, for the same reasons. The loss is for both higher and lower tones, but is generally typical of progressive perceptive deafness.

Between attacks the patient with Menière's disease often complains of tinnitus accompanied by a sense of fullness and pressure within the head. This symptom complex must be distinguished from the so-called symptomatic Menière's syndrome. This is a non-episodic syndrome consisting of vertigo, spontaneous nystagmus, and deafness, all of which are usually present at the same time. This type of syndrome is present in a wide variety of conditions. Those which relate specifically to the ear include neurofibroma of the basal coil of the cochlea, secretory and chronic adhesive otitis media, blockage of the eustachian tube, and cholesteoma.

Other Causes.—Other causes of deafness include allergy, which presents no difficulty unless it appears as an isolated phenomenon; streptomycin therapy, the effects of which on the eighth nerve are now so well established that little more than the history is required for diagnosis; hypothyroid disease, which is more likely to produce deafness if it is associated with feeblemindedness of mental dullness; and neoplasms, which vary in their effects according to their type and location.

SUMMARY AND CONCLUSIONS

The diagnosis as well as the differential diagnosis of deafness are complicated by the fact that only a few of the local and systemic processes which may cause it give rise to local objective signs in the living subject. In many instances diagnosis is a matter of inference. This perhaps explains why inexperienced otologists, as well as some experienced otologists, are inclined to believe that the investigation of deafness must consist of an elaborate routine which includes, among other items, the new testing technics developed since the science of electronics passed out of the theoretical stage.

This is a completely erroneous concept and a completely wrong course of action. By the use of such simple methods as careful history taking, a complete general and local examination, tuning fork tests and audiometric testing, any hearing impairment can be quickly demonstrated and can be classified as to type and also, with a fair degree of frequency, as to subtype. Hearing tests, as Guild[1] has usefully emphasized, are of value only insofar as they supplement data derived from the history and physical examination and as they confirm impressions already gained concerning which part or which parts of the auditory apparatus are functioning poorly. Only in the occasional case, Guild[2] continues, will the use of what Bezold, in 1896, called "time-robbing" testing methods add to what these simple and long established methods reveal.

If the experienced clinical otologist were to list the diagnostic methods available to him in the order of their usefulness, he would begin with a carefully taken history. If he had to discard the method of least usefulness, he would dispense with pure tone audiometry. Whatever value this presentation may have lies in its emphasis upon a return to these first principles. Every otologist knows them, but because of their simplicity he may be inclined to forget them or to undervalue them. The important consideration, as Guild[2] rightly concludes, is that, while he is examining his patients, he continues to think.

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MANAGEMENT OF THE THIRD STAGE OF LABOR*

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It is an interesting contrast that the placenta, so important an organ to the maintenance of intrauterine fetal life, should suddenly become so useless and potentially dangerous as soon as the baby is delivered. That this is true is attested by Greenhill's statement[5] that more women die as a result of the complications of the third stage of labor than of the accidents of the first two stages combined. Hemorrhage, notably postpartum hemorrhage (which includes the third stage), is one of the major causes of maternal death, and many cases of postpartum hemorrhage result from the mismanagement of the third stage of labor. Campbell[4] believes that hemorrhage is the first cause of maternal death, and that the real risk to the mother lies in the third stage. Among the controllable causes of postpartum hemorrhage, Eastman[8] lists mismanagement of the third stage of labor. Since this great hazard exists, the proper management of the placental stage is of paramount importance.

Anatomically, it is well to recall that the uterine sinuses are simply blood spaces, lined with a single layer of endothelium, which are interspersed through the muscular bundles and layers of the uterus. As the uterus is emptied there is contraction and retraction of the muscular elements with a consequent bending and constriction of these sinuses and a shutting-off of blood flow through them. Thus it is easily understood why bleeding from the sinuses not properly "clamped off" by the muscle can be as profuse as that seen in uterine atony.

Physiologically, the third stage of labor is divided into two phases:

(A) *Separation.*—This phase is spontaneous and occurs because of the decrease in area of the placental site which has begun in the late second stage. Leff[10] and Pastore,[13] among others, established the fact that the retroplacental hematoma is the result rather than the cause of placental separation, and that if it is large it indicates delayed expression. It is important to avoid manipulation of the uterus during the separation phase.

(B) *Expulsion.*—It is in this phase that prompt and active intervention is advocated in the interest of conservation of blood.

The combination of the policy of non-intervention in the phase of separation and immediate aid to the expulsive phase is considered appropriate management of the third stage.

"It is considered bad practice to drag out the body, except in very particular circumstances—the womb and abdominal muscles are sufficiently powerful for the object; and if it be permitted to come away slowly, we shall have a more complete contraction of the womb, and a more ready detachment and extrusion of the placenta."

Thus did Charles D. Meigs,[11] just one hundred years ago advise the slow delivery of the body of the child as a means of facilitating the third stage of labor. Dieckmann *et alii*,[7] in modern times, have shown that the slow and deliberately delayed delivery of the infant will definitely bring about a marked reduction in the incidence of postpartum hemorrhage due to uterine atony, and a more efficient separation phase of the third stage of labor. Their data showed that the prevention of postpartum hemorrhage begins with the proper conduct (slow delivery) of the terminal phase of the second stage of labor.

Since the classical signs of separation of the placenta, bleeding, rising of the uterus in the abdomen and advancement of the cord occur relatively long after the placenta has separated, Leff[10] advocated the immediate vaginal touch method of detection of placental separation and prompt expression of the placenta to prevent unnecessary blood loss.

Concerning the Credé method of placental expression, O'Driscoll and Quinn[12] believe that this classical maneuver is the hidden danger of the third stage. Greenhill[6] says that since the Credé maneuver has potential and actual dangers it should be relegated to the obstetric operations of the past. He also said, "Even the common method of early expression, using the corpus as a piston, is not without danger." This method is the so-called "modified Credé" widely used today. Brandt,[2] and Andrews[1] independently

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have shown the dangers of the forceful methods of placental expression, and have suggested a logical method which conserves maternal blood and avoids the possibility of inversion of the uterus. This has become known as the Brandt-Andrews method in which the separated placenta is expressed by upward pressure on the uterus, and downward pressure on the placenta, not the fundus, is made to complete the expression.

It is the conviction of the writer that holding the empty uterus up out of the pelvis until it is firmly contracted and keeping it out of the pelvis is an additional means of conserving blood which is the prime objective of a properly conducted third stage.

MATERIAL AND METHODS

Six hundred private patients comprise the two series here described. All cases involving abnormalities of the third stage and of the placenta were eliminated since it was desired to present a comparison of the two methods in the management of the normal patient. All babies delivered weighed 2500 grams or more and were in vertex presentation. Series A *(Table 1)* consisted of 300 cases in which there was no delay in the delivery of the baby, the abdominal detection of placental separation was practiced, and the conventional management of the third stage was used. Series B *(Table 1)* was made up of 300 cases in which slow delivery of the baby was used, together with the detection of placental separation by vaginal touch, and the Brandt-Andrews method of management of the third stage. In both series, the patients had either no analgesia, or demerol® and scopolamine analgesia, and were delivered without anesthesia, or with local or saddle block anesthesia. All patients were delivered in the University Hospital or the Hospital for the Women of Maryland. From *Table 1* it will be seen that the estimated average blood loss in Series A was 255 cc., while in Series B it was 115 cc. These estimates include episiotomy blood loss. *Table 1* also shows the reduction in the average duration of the third stage from 11 minutes in Series A to 4 minutes in Series B. Critics of this method are sure to point out that we have only estimated and not measured total blood loss for our statistics. We can only agree, but we also are convinced that we shall never return to the more common method of management of the third stage, since with the method of our choice we have constant control of the uterus and can readily detect abnormal behavior such as atony.

	Series A—300 Cases Delivery not delayed Abd. Det. Plac. Sep. Ordinary expression	Series B—300 Cases Delayed delivery Vag. Det. Plac. Sep. Brandt-Andrews expr.
Average blood loss Third stage (estimated)	255 cc.	115 cc.
Average duration Third stage	11 Minutes	4 Minutes

TABLE 1

Comparison of Total Blood Loss and Duration Third Stage

SUGGESTED METHOD OF MANAGEMENT OF THIRD STAGE

The following is a suggested method of management of the late second stage and the third stage of labor which has been proven clinically. It combines the essentials of the above-outlined procedures. The vertex is delivered slowly and deliberately, the upper respiratory passages being aspirated immediately *(Fig. 1)*. The shoulders and body are slowly delivered, the body being delivered

FIG. 1

Head delivered; aspiration.

as far as the umbilicus, where the process is stopped and a loop of cord pulled down so that it may be palpated *(Fig. 2)*. The arrest in delivery at this juncture is for at least 3 minutes or until definite uterine contractions are palpated. The latter usually begin within 2 minutes. During the delay, further aspiration is carried out and the baby nearly always will cry spontaneously. This delay has the further advantage of giving the baby additional blood as has been shown by Windle[14] and emphasized by Eastman[9] and others. The remainder of the baby is slowly delivered and the cord clamped and cut. Vaginal touch, as advocated by Leff[10] is immediately carried out to ascertain whether or not the placenta has separated *(Fig. 3)*. In most instances the placenta will be found separated and should be expressed according to the Brandt-Andrews technique which consists of upward stroking of the uterus *(Fig. 4)*, and downward pressure on the separated placenta *(Fig 5)* to complete

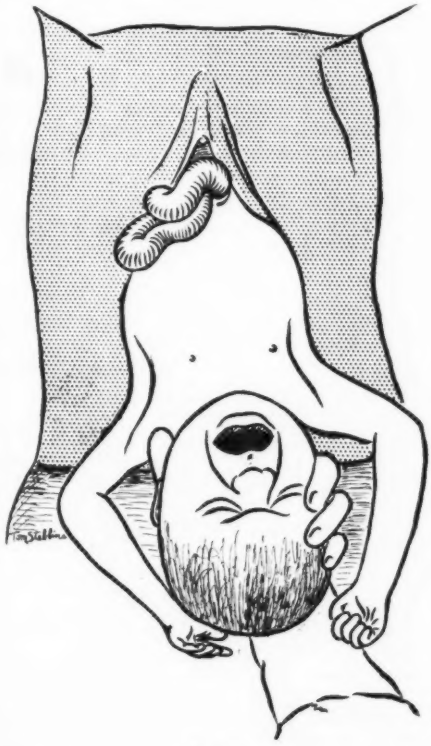

FIG. 2

Body delivered to umbilicus, and loop of cord pulled down.

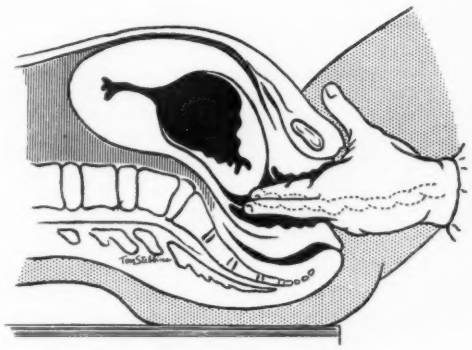

FIG. 3

Vaginal touch for detection of placental separation.

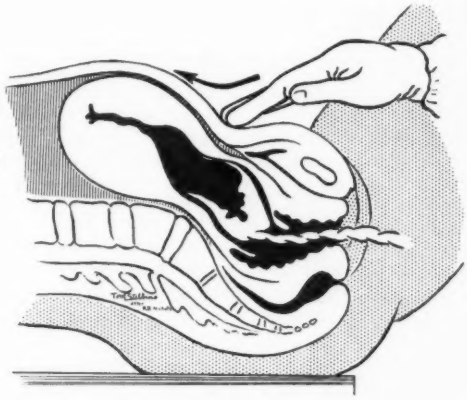

FIG. 4

Upward stroking of uterus with slight traction on cord.

FIG. 5

Downward pressure on the separated placenta.

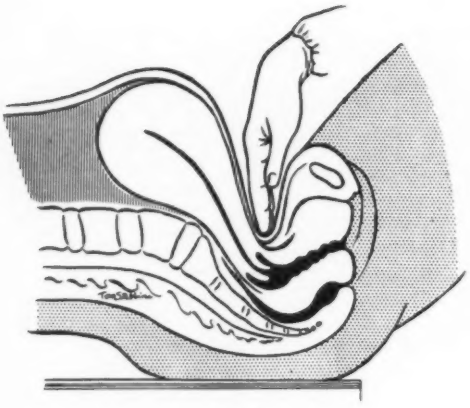

FIG. 6

Holding uterus up out of pelvis until firmly contracted.

its expression, the empty uterus being held out of the pelvis *(Fig. 6)*. The following is a description of the writer's oxytocic routine and not that of either hospital where these patients were delivered. Pitocin 0.5 cc. is given intravenously, and ergonovine, 0.2 mg., is administered intramuscularly. The uterus is held high until it is fully contracted. Bleeding during and after the third stage will be minimal. The placenta is then carefully inspected since this is an integral part of the conduct of the third stage. Cervical inspection is routinely carried out; and perineal repair is performed. A minimum period of 2 hours after the completion of the third stage should be devoted to careful observation of the uterus and the general condition of the patient.

If the patient is delivered in the home, the following technic may be used: slow delivery of the baby, the application of the Calkins'[3] sign for abdominal detection of placental separation by noting the change from discord to globular form of the uterus and the immediate application of the Brandt-Andrews expression.

SUMMARY

Adopting the principles enunciated by various authors and combining them into one philosophy of management of the third stage of labor, the following fundamentals are suggested:

(1) Slow delivery of the infant, which conserves maternal blood and provides additional blood for the baby.

(2) Vaginal touch for the detection of placental separation.

(3) Prompt application of the Brandt-Andrews method of placental expression.

(4) Holding the uterus up out of the pelvis until the oxytocics become effective and the uterus remains contracted. This procedure generally obviates the necessity for massage (with its attendant danger of displacing the placental clot) of the uterus after placental expulsion.

(5) Inspection of the cervix and examination of the placenta.

(6) Careful observation of the patient for a minimum period of 2 hours postpartum.

CONCLUSIONS

A more active management of the third stage of labor is advocated, consisting of slow delivery of the baby, immediate detection of placental separation by vaginal touch, prompt expression of the separated placenta by the Brandt-Andrews technique, and holding and keeping the uterus high out of the pelvis until it is completely contracted. By this method, a reduction in the estimated average blood loss in 300 cases managed by the described method as compared to the control series of 300 cases was 140 cc.; and in the same two series of cases, the average duration of the third stage of labor was reduced by 7 minutes.

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Six East Read Street.

DISCUSSION *(Abstract)*

Dr. H. Hoffman Groskloss, Miami, Fla.—One normally would not, at first thought, consider a discussion on the third stage of labor as particularly exciting, but one cannot easily dismiss the gravity of the proper conduct of this stage of labor when one learns that its mortality is greater than that of the first two stages combined. Postpartum hemorrhage is responsible for 4.9 per cent of maternal deaths; 16.0 per cent of these are due to retained secundines, 36.0 per cent to uterine atony, the others to causes to be referred to later. Postpartum hemorrhage occurs once in sixty deliveries and retention of tissue is met in one in five hundred of these incidents. Upon analysis of cases received, the chief errors of omission are: (1) delay or negligence in diagnosis and thereby, action to include blood replacement; (2) hesitating to explore the cervix and uterus for evidence of injury, presence of tissue, or uterine rupture; (3) underestimation of blood loss; (4) failure to realize limitations of oxytocics; (5) improper handling of the uterus before and after the expulsion of the placenta.

The uterus is too often stimulated or attempts at expulsion of the placenta are made before the complete separation of this organ. After intravenous ergonovine® is given with the delivery of the anterior shoulder appearing under the symphysis, followed by slow delivery of the child, the Calkins' sign of separation should be respected (expulsive action at this time is especially indicated to avoid retention when saddle block is used for anesthesia). If expulsion is delayed and one elects to repair the episiotomy while waiting, one must consider that serious occult bleeding into an atonic uterus may be taking place. With Calkins' sign the placenta is obtained by the Brandt-Andrews method as shown by Dr. Savage. Recently, Picton has advanced the idea of traction on the cord while holding the uterus well out of the pelvis. The uterus is then well massaged out of the pelvis until firmly contracted.

If the uterus is behaving normally and bleeding continues, and secundine retention and cervical bleeding are eliminated, packing may be resorted to, but it is only to be attempted once. One should be warned against the apparent security of packing, and remember especially that ten yards of packing may absorb a full liter of blood. Preferably to repacking one might resort to a hot caustic intrauterine douche. If these measures fail, blood replacement is mandatory, followed by Kerwin clamping of the uterine arteries, or vaginal or abdominal hysterectomy. It may be well to suggest that being confronted by the signs and symptoms of shock, pulmonary or cardiac complications should not be overlooked. The failure of the first packing would suggest the presence of cervical injury, uterine atony, Couvelaire or ruptured uterus, open low segment sinuses or blood dyscrasia.

Dr. Donald G. Tollefson, Los Angeles, Calif.—In California they say we do not see many placentas coming through the vagina because we deliver all the patients from above. This is about the first time I have heard anyone with courage to say that he does a vaginal examination to find that the placenta is separated.

The signs in the text books about the rise of the uterus and the descent of the cord and the gush of blood, I think, should be revised, because that is not the way to determine that the placenta is separated. These signs are not practical. In California we feel that the vagina can be invaded after the baby has been delivered, and even before, to determine exactly what is happening.

We have been repairing many of these cervices, and we are very satisfied with the results.

Dr. H. J. Langston, Danville, Va.—Speaking of postpartum hemorrhage, Dr. Hubert Royster, of Raleigh, North Carolina, when he was interning at Pittsburgh many years ago was taking care of a case for some one of the visiting doctors. It was a spontaneous delivery, and afterwards the patient looked as if she were going to bleed to death. Dr. Royster seized upon a piece of ice and stuck it up in the uterine cavity and it stopped the bleeding. He was very much pleased with the results. So the next morning when the visiting obstetrician came by, Dr. Royster told him about it, and the visiting staff man said, "Next time, sterilize the ice."

This third stage of labor has been more alarming to me in the past thirty years than any other stage.

In the second stage of labor, when the patient is taken to the delivery room, I put a catheter in the bladder and let it stay there until we have finished our work. I leave the catheter in the bladder.

After the second stage of labor, I leave the baby attached to the cord and put it up on the abdomen of the mother. I have one of the nurses support the baby by its feet.

Now, nurses always want to grab something, doctors always want to grab something, and they are always grabbing the uterus. Take your hands away from the uterus, and keep them away.

As to Dr. Andrews' method of handling the uterus, I have been using it about twenty-nine years. It works.

For a number of years I have been repairing the cervix when it is torn, and many cervices are torn in spontaneous delivery. This can be done without too much trouble, before repairing the perineum.

While the placenta is separating, if I have much of a tear, I repair that before I get the placenta out. I don't know that that has any advantage except that it saves time.

Dr. H. H. Ware, Jr., Richmond, Va.—I agree with Dr. Savage generally in the way he handles the third stage of labor.

Sometimes we fail to realize that in many cases, hemorrhage in the third stage of labor results from mismanagement of the early stages of labor. Patients with inertia and prolonged labor often become exhausted, dehydrated, and need fluids, rest, and sedation. In some sections of the country where many referred patients are admitted to the hospitals without any prenatal care they are frequently poorly nourished and anemic. They come in with a marked anemia, and they are poor risks and go into shock easily. Often their hemoglobin and red cell count will be far below the normal level, and the loss of a small amount of blood may be fatal for these patients.

It is important to correct anemia during the prenatal period. The pregnant woman should eat a well balanced diet and we must try to improve her health and nutrition and bring her to the optimum state of health before the onset of labor.

It has been our belief that it is best to deliver the placenta before we repair the perineum. It is difficult to do a good repair when there is considerable bleeding from the uterus which may make it difficult to approximate the tissue correctly. Deep anesthesia predisposes to relaxation of the uterus and excess bleeding. If the placenta is left in and it becomes necessary to remove it after the perineum has been repaired, frequently the manipulation breaks down the perineorrhaphy.

We usually give to the mother 0.5 cc. of pitocin® intramuscularly after the baby has been delivered and then 1 cc. of ergotrate intramuscularly after the placenta has been expelled.

Patients who receive analgesia during labor and inhalation anesthesia at the time of delivery are likely to get some paralysis of the uterus, which, of course, will prolong the period of separation of the placenta and may increase the amount of uterine bleeding.

I feel that if the patient is delivered under inhalation anesthesia, it is a good idea for the anesthetist to empty the bag of all of the anesthetic agent, whatever it is, and give the mother straight oxygen for about two minutes as the fetal head comes out over the perineum. This will give the baby a good pink color when it is just born and it will breathe much more quickly. It will also give the uterus a better tone, decrease the amount of uterine bleeding, and shorten the third stage of labor.

Dr. Matthew W. Weis, St. Louis, Mo.—I rise not to defend the douche, but to explain it. A douche was developed by Washington University in St. Louis some years ago, not to prevent or to control postpartum bleeding, but to be used after criminal or infected abortions. Later it was employed in certain cases of uterine bleeding after the third stage, but it originally was not designed for that purpose. It is a potassium permanganate douche. I think there is a little sulphuric acid in it, and I do not know of anybody who has used it recently.

Dr. W. E. Hoffman, Charleston, W. Va.—At Polyclinic in New York, it is the practice to lift up the anterior lip of the cervix to get the placenta out. This relieves much of the hemorrhage that follows in the third stage.

I have my nurse push on the fundus, and I lift up the anterior lip of the cervix. If then I put my finger in the vagina, I will not infect the uterus but can push the placenta out.

I read an article in the *American Journal of Obstetrics and Gynecology* about putting ice in the vagina in postpartum hemorrhage. About ten or fifteen years ago, I used it in one case. She went home in five days. In ten days she was in the hospital again with the nicest tetanus you have ever seen. She could not get her jaws open.

I called in internists and she recovered. I had a coke with her the other day.

Dr. Savage (closing).—We used Calkins' sign entirely before adopting the method of detection of placental separation by vaginal touch. An obese abdomen and obstetrical drapes often interfere with the application of Calkins' method. You will recall that we did recommend the use of Calkins' abdominal detection of placental separation in home deliveries where the technic and surroundings were not ideal.

We repair the episiotomy only after the placenta is expressed. To prevent further blood loss from that source distinct bleeding vessels are ligated and pressure with sterile gauze is made on the wound if there is any period of delay.

Since using the method advocated here, and long before that, we discontinued packing the uterus and using intrauterine douches.

We have the definite clinical impression that with saddle block, or any other form of regional anesthesia, the incidence of postpartum hemorrhage is greatly reduced.

We only use pitocin® during the second stage of labor in the form of a dilute pitocin® drip, primarily in cases of uterine inertia, and then only when no contraindication for its use exists. With the use of pitocin® drip we have observed no increase in the incidence of abruptio placentae or postpartum hemorrhage.

We have had no experience with leaving a catheter in the bladder.

Dr. Langston also pointed out that he allowed the placental circulation to remain intact by not clamping the cord at once, and he places the baby across the mother's abdomen. Such a maneuver interferes with the application of Calkins' sign. However, if one uses vaginal touch to detect placental separation, much time and therefore blood will be saved. We allow the baby to receive additional blood during the purposely delayed second stage of labor during which the delivery is stopped at the umbilicus.

We regret that we did not know of Dr. Langston's prior claim to the principle of the Brandt-Andrews' technic.

No patient in this series received inhalation or intravenous anesthesia.

LONG ACTING INTERCOSTAL NERVE BLOCK*

A SAFER METHOD OF ANESTHESIA FOR ABDOMINAL SURGERY

By ROBERT W. BARTLETT, M.D., F.A.C.S.†
and
DOUGLAS W. EASTWOOD, M.D.‡
St. Louis, Missouri

Some years ago it became apparent that there must be a safer method of anesthesia than deep ether or spinal for upper abdominal surgery in seriously debilitated individuals. As a result of considerable study and clinical research, the method of bilateral intercostal nerve block was evolved and introduced by R. W. B.[1] in 1940. This consisted of bilateral blocking with procaine of the sixth through the eleventh intercostal nerves in the mid axillary line in combination with a very light general anesthesia, the latter providing anesthesia of the visceral peritoneum so as to permit packing off of intestines, whereas the former resulted in complete relaxation of the recti and flat muscles plus anesthesia of the parietal peritoneum.

The end result was excellent exposure of intra-abdominal structures, easy accurate closure of the incision, and, finally, a patient who was awake soon after the conclusion of the operation. In other words, one was able to enjoy all of the advantages of spinal or full ether anesthesia without any of the dangers associated with them.

Shortly after the introduction of this method of anesthesia we realized the possible advantages of a long acting intercostal nerve block. It was logical to suppose that a patient with a relatively painless incision would be free of the usual splinting of the diaphragm which results in limitation of respiratory excursions and coughing, resulting at times in the development of atelectasis and subsequent pneumonia. At a somewhat later date, the advantages of free early ambulation associated with a relatively painless incision were appreciated.

As early as 1941, we and others[2,3,4] began experiments with intercostal nerve block, employing the oily solutions then available for prolonged anesthetic action, but found that the diffusion of the anesthetic agent from such solutions was limited, thus giving results which were so spotty as to be impractical for routine use.

In 1951, efocaine,®* a new long acting anesthetic agent, was brought to our attention and furnished to us for clinical trial. Efocaine consists of two anesthetic agents, procaine and butyl-amino benzoate, in a nontoxic organic solvent base. These agents are precipitated out at the site of injection by coming into contact with tissue fluids and their subsequent slow reabsorption is said to provide the anesthesia or hypesthesia usually lasting from 7 to 14 days.

The technic of block, as originally outlined,[1] has been modified but little during the past twelve years and will be reviewed briefly. The patient is placed on the operating table with both arms abducted on boards, following which light pentothal® and nitrous oxide in combination, or light cyclopropane anesthesia is induced.

The skin of the lateral chest wall is suitably prepared bilaterally, then the surgeon or the physician-anesthetist, having donned a pair of sterile gloves, carries out the block, employing a standard 10 cc. syringe and No. 18 caliber needle with a point sufficiently blunt so that its passage through a fascia layer can be appreciated. The syringe is filled from the rubber-stoppered 20 cc.bottle of efocaine® solution, then the sixth through the eleventh intercostal nerves are blocked bilaterally in the mid axillary line. One palpates the lower border of a rib, then the needle is passed through the skin and fat down to it; following this the needle is allowed to slip off the lower border of the rib and in so doing it is easily felt to pass through the fascia covering the external intercostal muscle. The direction of the needle is slightly upward into the groove beneath the rib. The point of the needle now lies between the two intercostal muscles where the intercostal vessels and nerve are situated. After drawing back on the plunger to prevent intravascular injection, a deposit of 1 cc. of the anesthetic solution is made. With this dosage there have

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*Efocaine®, E. Fougera & Co., Inc., New York, N. Y.

been a few instances of unexplained local pain postoperatively at the site of injection such as invariably occurred in our earliest cases when a 3 cc. dosage was employed. If one person performs the bilateral block, about the same time is required as for administering a spinal anesthesia; however, if two persons each do a side simultaneously, a matter of only two or three minutes is involved in the procedure.

As mentioned earlier, splendid relaxation of the upper abdominal muscles is present by the time the skin and fat have been incised so that excellent exposure of deep structures, such as the common duct, is easily obtained; likewise, accurate closure of the wound is possible in such a subject. If, occasionally, a patient pushes intestines into the wound, it means that the general anesthesia is so light that he is feeling pain due to packing or handling of the visceral peritoneum; the pushing is probably due to the action of the lower abdominal musculature plus the diaphragm and this situation can be corrected in thirty seconds by the injection of an additional small amount of pentothal.®

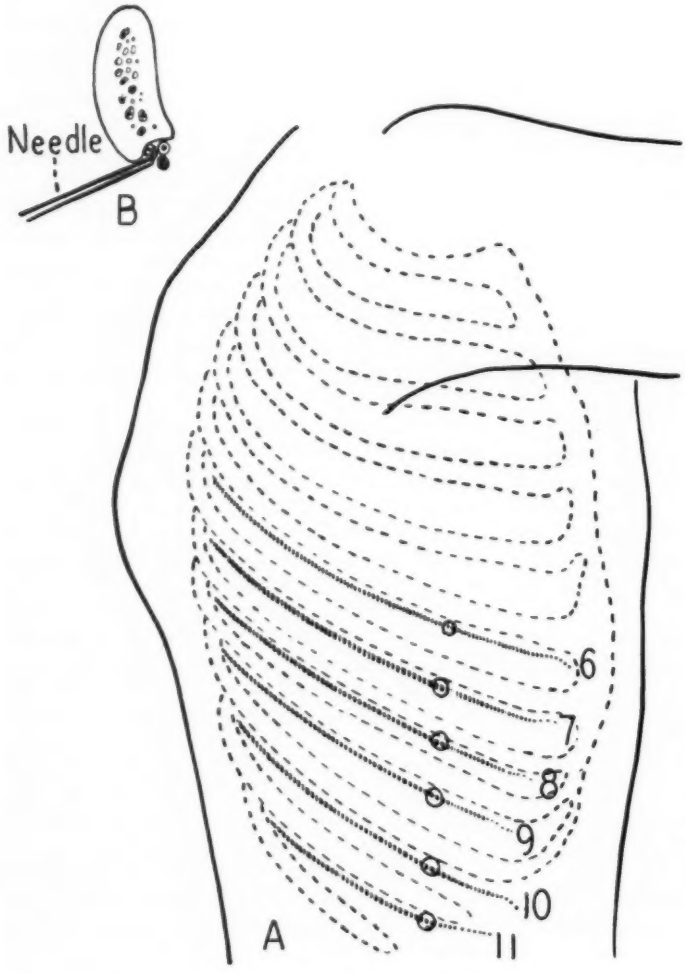

FIG. 1

(A) Sites of injection of sixth, seventh, eighth, ninth, tenth and eleventh intercostal nerves in mid-axillary line.

(B) Nerve lying in costal groove at inferior border of rib between external and internal intercostal muscles.

Our method is not to be confused with the procedure carried out by Puderbach and Shaftel[5] and also others who block the intercostal nerves for pain relief according to our original technic,[1] but only at the conclusion of the operation which has been carried out under one of the conventional forms of anesthesia for major surgery. It is the usual deep anesthesia and its possible sequelae that we seek to avoid, especially in the bad risk patients.

The reports of Weinberg[6] would indicate that local tissue damage resulting from injection of the drug is probably minimal. One should take the same precautions as with ordinary procaine against intravascular injection of the solution.

Our series of cases anesthetized by this method totals 75 up to the present time and consists for the large part of the definitely major types of cases tending to gravitate to university hospitals. The method is employed equally satisfactorily for intra-abdominal or abdominal wall lesions, also for thoraco-abdominal incisions. As concerns postoperative complications attributable to the method of anesthesia, a case of bilateral pneumothorax occurred early in the series which was due to a technical error and which was quickly relieved. There has been no clinically recognizable atelectasis nor pneumonia though we realize that such instances will occur from time to time with increasing experience. In addition, there has been no instance of thrombophlebitis, embolism nor wound separation to date. It might be added that almost all of the major incisions have been closed with figure-of-8 buried interrupted fine stainless steel sutures.

Approximately 25 per cent of the cases have been from the ward services and 75 per cent from the private service of R. W. B. Of the two deaths in the series, one occurred in a ward case following subtotal gastrectomy in a patient with both pre- and postoperative auricular fibrillation who went into acute failure one week postoperatively. The one death on the private service occurred five weeks postoperatively as a result of multiple intra-abdominal complications following reconstruction of a common duct, 1½ inches of which had been resected at the time of cholecystectomy elsewhere some weeks previously with a resulting complete external biliary fistula.

Under this plan of anesthesia, the patient is awake at or soon after the conclusion of the

TWENTY-FIVE CONSECUTIVE LAPAROTOMIES SINCE INSTRUCTING NURSING PERSONNEL REGARDING MORPHINE REQUIREMENTS

Name	Operation	Complications	Results	Postop. Opiates
C.P.	Exploration common duct	None	Recovery	None
J.S.	Gastric resection	None	Recovery	None
I.M.	Umbilical herniorrhaphy, large	None	Recovery	Demerol® 100 mg. x3
C.D.	Gastric resection	None	Recovery	None
E.M.	Hemicolectomy, right	None	Recovery	None
R.B.	Gastroenterostomy; vagotomy	None	Recovery	None
M.A.	Ventral herniorrhaphy, large	None	Recovery	None
O.L.	Laparotomy for intestinal obstruction	None	Recovery	None
B.O.	Exploration common duct	None	Recovery	None
R.L.	Gastric resection	None	Recovery	None
V.J.	Gastric resection	None	Recovery	None
R.C.	Gastroenterostomy; vagotomy	None	Recovery	None
B.B.	Gastric resection	None	Recovery	Moph. gr. 1/6 x1
J.N.	Gastric resection	None	Recovery	None
T.V.	Gastric resection	None	Recovery	None
J.B.	Gastric resection, third operation	None	Recovery	Demerol® 50 mg. x4
O.H.	Exploration common duct	None	Recovery	Morph. gr. 1/8 x1
D.W.	Cholecystectomy	None	Recovery	Morph. gr. 1/6 x4
B.R.	Reconstruction common duct	Multiple Abd.	Died	None
T.H.	Gastric resection	None	Recovery	None
E.E.	Cholecystectomy	None	Recovery	None
E.T.	Gastric resection	None	Recovery	Morph. gr. 1/6 x1
M.W.	Drainage lesser sac abscess	None	Recovery	None
H.T.	Vagotomy for marginal ulcer	None	Recovery	Morph. gr. 1/6 x1
B.B.	Exploration common duct	None	Recovery	Morph. gr. 1/6 x2

TABLE 1

operation; he is more active in bed during the first few hours postoperative and he gets up sooner than the average patient. Deep breathing and coughing without undue discomfort are possible and it is our impression that catheterization and enemas are less frequently required. The hypesthesia or anesthesia to pin-prick in the region of the incision lasts ordinarily from 7 to 14 days, though occasionally longer, and there is an obvious marked decrease in the number of injections of postoperative morphine required, thus making for a more active and alert patient requiring less nursing care. It should be mentioned at this time that deep visceral discomfort and "gas pains" which sometimes occur about the second postoperative day must be differentiated from incisional pain and are in no way influenced by the intercostal nerve block. Since we have instructed the nursing staff that morphine may at times contribute to the production of nausea and distension and that it is required for real pain only, while sodium luminal® grains 2 hypodermically is adequate for restlessness or general discomfort, our records show that in the most recent 25 cases, 17 have required no postoperative opiate, 4 have received morphine grains 1/6 one time only, and 4 have required morphine grains 1/6 from 2 to 4 times.

In recent weeks we have extended the scope of the method to include operations in the lower abdomen and pelvis by adding injection of the twelfth intercostal nerves to the routine technic of the sixth through the eleventh. While the usual blocking of intercostal nerves is ordinarily quite simple, the twelfth will be found to be a bit more difficult since the rib is very short, making it necessary that the patient be rolled up about 45 degrees off his back while this nerve is being injected.

Excellent muscular relaxation is obtained all the way down to the pubis, but sensory anesthesia stops about 2 inches above that level. While we have as yet no definite data on the subject, it is our impression that an incision sensitive at its lower 2 or 3 inches is probably less painful than one sensitive for 8 or 10 inches. We have carried out to date with considerable satisfaction eleven operations in this latter category, including right and left hemicolectomy, right rectus appendectomy, hysterectomy and suprapubic removal of a large urinary bladder calculus. At a future date a more detailed communication will be forthcoming concerning this latter phase of the problem.

The anesthetic agents currently available for prolonged action at times exhibit undesirable features which will have to be improved for routine use; however, we believe that it has been clearly shown that the method of long acting intercostal nerve block adds greatly to the safety and comfort of the patient and if one has any doubts as to the accuracy of the latter statement, he need only ask a patient to compare an old with a recent surgical experience under this method.

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ACUTE PANCREATITIS: DIAGNOSIS AND TREATMENT*

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The discussion to follow is based in large part upon a review of the findings in thirty-three patients with acute pancreatitis studied at the Graduate Hospital (350 beds) over a ten-year period (1941-1951). Unfortunately, all cases with this diagnosis could not be used because of inadequate objective data in some of the charts of patients seen in the earlier years. Six charts were not available at the

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time of compilation. Although an average of four patients were admitted annually during this period of study, in recent years the incidence has been increasing progressively because of a greater awareness and the more frequent employment of the pancreatic serum enzyme tests.

The age of patients in this series ranged from twenty-eight to seventy-eight years. The average age was forty-seven years. Twenty were males, thirteen females. Ten were negro, twenty-three white. Considering the ratio of negro to white admissions to the wards of the Graduate Hospital, this does not represent a racial predisposition or increased incidence of acute pancreatitis among negroes.

Possible Etiologic Factors.—Table 1 indicates those important factors which should be given consideration in the taking of a history in the patient suspected of having acute pancreatitis. An analysis of these cases confirms previous impressions as well as the observations of others. Approximately one in three patients had had previous attacks of acute pancreatitis. A history of gallbladder disease was noted in one-third of the patients. Unfortunately data was not obtained in some instances concerning addiction to alcohol or upon excessive imbibance of alcohol just prior to the attack of acute pancreatitis. Nevertheless, it is clear that alcohol plays a prominent role as a precipitating cause of attacks in a considerable number of patients. A history of excessive imbibance of alcohol should suggest the possibility of acute pancreatitis in any patient with severe abdominal symptoms.

The occurrence of biliary tract disease and of over-indulgence in alcohol in relation to the development of acute pancreatitis is further correlated in *Table 2*. In this table the actual incidence of biliary tract disease is given, based upon both history and subsequent study. We believe it is fair to state that in approximately 90 per cent of our cases of acute pancreatitis, biliary tract disease or overindulgence in alcohol were probably concerned with the occurrence of the pancreatitis.

Classification: Necrosis versus Edema.—It is realized that there are all grades of acute pancreatitis varying from mild edema to massive necrosis of the entire gland. Nevertheless, for clinical purposes it is serviceable to classify cases into two main groups: (1) those with edema and little or no necrosis and (2) those in which considerable actual necrosis of pancreatic tissue has occurred. In *Table 3* our cases are so classified. In eleven instances the diagnosis of necrosis or edema was proved by operation. In the remainder of the cases, the designation of necrosis or edema was based on criteria which will be discussed later. It may be stated now that clinical and laboratory data available during the first twenty-four hours of observation rarely make it possible to distinguish with certainty between necrosis and edema. Necrosis was noted at subsequent operation in two patients, who on admission were presumed to be suffering

ACUTE PANCREATITIS, 33 CASES
HISTORY: POSSIBLE ETIOLOGIC FACTORS

	No.	Per Cent
Antecedent attacks of probable or proved pancreatitis	11	33
Previous attacks gallbladder disease (previous operations, 4)	10	30
Previous duodenal ulcer perforation	1	—
Alcoholics (7 of 16 in which data available)	—	44
Alcohol prior to attack (8 of 17)	8	47

TABLE 1

ACUTE PANCREATITIS, 33 CASES
PATHOGENETIC CORRELATION

	M.	F.	Total No.	Per Cent
Biliary tract disease	8	10	18	55
Alcohol (relation to attack)	6	2	8	24*

*Data incomplete, Incidence probably greater

TABLE 2

ACUTE PANCREATITIS, 33 CASES
CLASSIFICATION: NECROSIS VERSUS EDEMA

	Proved*	Probable	Total	Per Cent
Necrosis	8†	4	12	36
Edema	3‡	18	21	64

*Proved by operation.

†Five operations performed on the basis of a wrong diagnosis, three of which were performed prior to era of enzyme determinations. Three operations for biliary tract disease after subsidence of acute pancreatitis.

‡Three operations for biliary tract disease after subsidence of acute pancreatitis.

TABLE 3

only with so-called edema of the pancreas in association with biliary tract disease. Slightly over one-third of our patients were thought to have had acute necrosis of the pancreas.

Clinical Features.—A review of these cases confirms our previous impression that it is rarely possible to make a definite diagnosis of acute pancreatitis solely by bedside examination. Almost all experienced clinicians admit that acute pancreatitis follows no consistent symptom pattern, that it masquerades in many different guises (Bockus and Raffensperger[1]). It is not our purpose in this presentation to review the salient clinical configurations encountered.

Value of X-Ray Study.—The principal value of roentgen study in these patients is tabulated in *Table 4*. The examination was utilized in a number of instances to exclude pneumoperitoneum, when free perforation of peptic ulcer was under consideration. We have never encountered pneumoperitoneum in association with primary acute pancreatitis. Chest signs, usually basal, were noted on physical examination in six patients and roentgen abnormalities were noted bilaterally in three of them, and at the right lung base in one. Symptoms and signs suggestive of pneumonitis or pleuritis occur with sufficient frequency in pancreatitis to cause difficulty in the differential diagnosis in some instances.

Small bowel obstruction is mimicked by acute pancreatitis in other patients, so that plain film of the abdomen may be utilized in an effort to distinguish between apparent ileus due to acute pancreatitis and actual bowel obstruction. This occurred in four of these patients. Gallstones were noted in two patients and suspected in a third on the plain abdominal film. In this series pancreatic calculi were noted in only one patient.

ACUTE PANCREATITIS, 55 CASES
VALUE OF X-RAY

Plain Film	(1)	Excludes pneumoperitoneum	
	(2)	Signs at lung base	3
	(3)	Differentiation ileus versus obstruction	4
	(4)	Gallstones	2
	(5)	Pancreatic calculi	1
Barium Enema		(emergency) to exclude obstruction sigmoid	1
Barium Meal		not done in acute fulminant attack	

TABLE 4

An emergency barium enema was performed at the instigation of the admitting officer in a patient whose symptom configuration and objective findings suggested volvulus of the sigmoid. In the fulminating types of acute pancreatitis, it is often not possible and usually unwise to carry out barium meal study. In other instances, when the disease is not suspected, or when studies are carried out after subsidence of the more acute phase of the illness, roentgen abnormality of the stomach, duodenum and jejunum have been recorded. These changes are quite similar to those which have been described in some cases of carcinoma of the pancreas. Bove, Cintra and Ferreira Santos[2] have reported a rather extensive experience with barium meal studies in acute pancreatitis (milder cases of edema). They mention the frequent occurrence of: (1) anterior-cephalad displacement of the stomach, (2) straightening of the greater curvature of the stomach, (3) atonic stasis in the duodenum and upper jejunum, (4) widening of the duodenal loop, and (5) downward displacement and distention of the duodeno-jejunal angle.

SERUM ENZYMES IN DIAGNOSIS

Methods and Normal Values.—In testing for amylase we employ the Somogyi technique; the amylolytic activity of the serum hydrolyzes starch to a reducing sugar. Serum amylase values average 50-60 mg. per cent in our laboratory. Although 120 mg. per cent is the upper limit of normal, values from 100 to 120 mg. per cent are considered borderline and interpreted in the light of previous determinations (if available) on any given patient. The values vary from laboratory to laboratory depending upon the substrate used. For this reason, it is well to consider abnormal values from various clinics in terms of so many times the top normal for reasons to be discussed.

For lipase determinations we employ the Cherry and Crandall modification of Loevenhart. Lipase in the serum liberates fatty acids from the olive oil substrate. Twenty-four-hour incubation is required; titration is with

sodium hydroxide. Serum lipase values average 0.3-0.4 cc.; the upper limit of normal in our laboratory is 1.2 cc. N/20 sodium hydroxide.

Relative Value of Amylase and Lipase Tests.—The amylase test has greatest value in the patient admitted as an acute pancreatitis suspect, principally because the reading is available in one hour. A twenty-four-hour incubation is required for the lipase reading. Furthermore, oftentimes the amylase concentration of the serum becomes abnormally high sooner after onset of the attack than does the concentration of lipase. However, it should be borne in mind that, not only does the elevation in lipase concentration reach its peak later than that of the amylase, but often it remains increased for a longer period. For this reason it is advisable to perform both tests. This is particularly important when the patient is not seen until two days or more after the onset of an attack, or if the precise time of onset cannot be clearly established.

Initial Amylase Values and Degree of Inflammation.—We have had the clinical impression that very high concentrations of amylase were more suggestive of the presence of necrosis rather than of acute edema of the pancreas. A review of these cases would suggest that the height of rise in both amylase and lipase values cannot be used in estimating the degree of inflammation or the presence or absence of necrosis. In *Table 5*, the amylase and lipase values are recorded in nine patients within seventy-two hours of onset of illness. The greatest concentration of both amylase and lipase in the serum was obtained in a proven case of edema of the pancreas.

ACUTE PANCREATITIS
NINE PATIENTS, FIRST ATTACK

Edema	Amylase	Lipase
Proven	2210	16.1
	844	—
Probable	825	12.7
Necrosis		
Proven	1417	14.3
	934	4.5
	1146	10.0
	412	—
Probable	1068	3.7
	1051	—

TABLE 5

Do Normal Values Occur in Acute Pancreatitis?—If the determination is carried out within seventy-two hours of the onset of symptoms during a first attack of proved pancreatitis, the serum enzyme values will be elevated almost always. One might anticipate normal values at times if the determinations are not performed early after the onset of an attack. In one of these patients in whom pancreatitis was presumed to have developed several days after the onset of acute cholecystitis, the serum enzyme values remained normal for three or four days initially. After a transient elevation for one or two days, the serum enzymes again returned to normal range for three days, only to rise again with considerable fluctuation that persisted for an additional two weeks. At the time of cholecystectomy on the twenty-fifth day of illness, edema of the pancreas was encountered as well as cholelithiasis and subsiding inflammation of the gallbladder.

Theoretically normal values may be obtained in association with acute pancreatitis if: (1) the determinations are carried out after three days when the values may have returned to normal, or (2) if previous attacks have destroyed the acinar cells of the pancreas.

Duration of Rise in Serum Enzymes and the Clinical Course.—Ordinarily both the serum amylase and lipase values have returned to normal within one week in patients who are experiencing the first attack of acute edema of the pancreas. This observation is based upon a study of those patients who have been on rigid medical management. If under such circumstances, increased concentrations of enzymes in the serum persist longer than one week, one may suspect that some degree of pancreatic necrosis is present. Although persistent elevations were present in all of our proved cases of necrosis, the enzyme values remained increased beyond one week in two of four cases of edema at the time of surgery.

Serum Enzyme Concentrations in Recurrent Attacks.—A review of the literature suggests that in some patients who have experienced an attack of severe necrosis, subsequent attacks may not give rise to such strik-

ing elevations in the serum enzyme levels. Our experience is too limited to confirm or refute this observation since only one patient in this series with necrosis was re-admitted for recurrent pancreatitis, and there was no significant difference in the enzyme values. Perhaps if a sufficient number of patients could be observed who suffer repeated attacks, it would be found that the greater the number of attacks, the less will be the rise in enzyme values. Occasionally, in some patients with chronic relapsing pancreatitis, severe pain attacks may provoke no increase in enzyme concentrations. Under these circumstances, it may be assumed that the acinar architecture has been destroyed.

FALSE POSITIVE SERUM ENZYME TESTS IN THE ABSENCE OF ACUTE PRIMARY PANCREATITIS

(1) *False Positives Following Administration of Opiates.*—Administration of morphine and allied compounds has been followed by increased cholecochal pressure attributed to spasm of the biliary duodenal musculature. This mechanism has been employed in attempting to establish the diagnosis of biliary dyssynergia (Butsch, McGowan and Walters[4]). Burke[3] and his associates reported the occurrence of increased serum amylase concentration in three normal persons following injection of morphine sulfate hypodermically. Gross, *et alli*,[6] obtained elevations in serum amylase and/or lipase within four hours following the hypodermic injection of two grains of codeine in five patients with biliary dyskinseia (post-cholecystectomy syndrome), and in one patient with duodenal ulcer penetrating into the head of the pancreas.

Since the patients with acute pancreatitis may have had an injection of opiate within 24 hours prior to hospital admission, it is of clinical importance to know whether such injections may in themselves cause a rise in amylase and/or lipase in patients without pancreatitis. One of us (Bogoch) has injected morphine hypodermically in thirty-three patients (36 tests) without evidence of pancreatic disease. Amylase and lipase determinations were made at 1.5, 2.5, 4 to 5 hours and 24 hours after injection. Rises in serum amylase above our normal range were noted in nine tests (25 per cent) from 2.5 to 5 hours after injection. In these cases, the range for serum amylase was 122-786 mg. per cent (average 283 mg. per cent). In three tests (2 of 16 patients), the values for amylase remained above normal at 24 hours after injection, that is, 151, 171, and 139 mg. per cent (22 per cent of patients with an elevation at 2.5 to 5 hours, or 13 per cent of those with 24 hour determinations).

In ten tests (28 per cent) in eight patients, pathologic elevations in serum lipase (above 1.5 cc.) were found at 2.5 to 5 hours. These values ranged from 1.6 cc. to 9.4 cc. (average 3.9 cc.). Nine patients had serum lipase determinations 24 hours after morphine injection. Five of these patients had elevations at 2.5 to 5 hours after injection; and of these one had an abnormal value persisting to 24 hours (1.8 mg. per cent).

This study indicates that occasionally in patients in whom there is no clinical evidence of pancreatic disease, one may obtain elevations in serum amylase or lipase within the range of values obtained in primary acute pancreatitis, if the blood is withdrawn within five hours after injection of morphine. Furthermore, slight increases in enzyme concentration may persist as long as 24 hours. This possibility must be considered when one is attempting to interpret the results of the serum enzyme tests in patients who have received injection of an opiate.

(2) *Hyperenzymemia in Extrapancreatic Disease.*—It is important to remember that increased pancreatic serum enzyme values are not always the result of acute inflammation of the pancreas. Raffensperger[7] reported the occurrence of pancreatic hyperenzymemia in twenty patients with extrapancreatic disease observed during one year at the Graduate Hospital *(Table 6)*. It should be noted that

HYPERENZYMEMIA IN EXTRAPANCREATIC DISEASE

	Number	High Amylase Value (mg.)	High Lipase Value (cc.)
Peptic ulcer perforation	5	326	6.2
Intestinal obstruction	4	343	2.5
Peritonitis	8	405	5.2
Uremia	3	326	5.9

TABLE 6

in acute abdominal conditions which may simulate acute pancreatitis clinically, he failed to obtain a serum amylase value which approached or exceeded five times our top normal value. During the past year, a patient with uremia had a serum amylase value of 586 mg. per cent. This is the highest determination which we have obtained for amylase in abdominal disease not of pancreatic origin. In the differential diagnosis of the so-called "acute abdominal emergencies," we believe it is safe to make a diagnosis of primary acute pancreatitis, if the serum amylase concentration exceeds five times the top normal value, but such magnitude of elevation is by no means always encountered nor essential to the diagnosis.

Acute Pancreatitis with Acute Biliary Tract Disease.—Since the advent of the pancreatic serum enzyme tests, an increased incidence of acute pancreatitis in association with both acute cholecystitis and biliary colic has been noted. We believe that immediate operation is contraindicated in the presence of acute pancreatitis, unless clear-cut indications render operation imperative. For some years, serum enzyme tests have been employed in all patients admitted to our service who have symptoms and signs suggesting acute cholecystitis or biliary colic. If this policy is not adopted, one certainly should do serum enzyme tests when an increased likelihood of acute pancreatitis is suggested by: (1) pain of greater intensity than is anticipated in attacks of "pure biliary colic," particularly if its greatest intensity remains in the epigastrium or radiates to the left upper abdomen or to the region of the angle of the left scapula; (2) association of shock-like state; (3) striking constitutional reaction, greater elevation in temperature and higher leukocytosis and (4) failure to obtain lasting relief from opiate injection. If hyperenzymemia is noted, our policy is to delay operation for the biliary tract disease until the pancreas has had time to "cool off," as indicated by a return to normal of the enzyme values. Hyperbilirubinemia is often concomitantly present in such instances and ofttimes the concentration of serum bilirubin will return to normal concomitantly. If this fortunate happenstance does take place, the patient is a better operative risk and definitive surgery is more easily accomplished. Obviously, the acuity of the biliary lesion demands earlier operation in some instances. Acute pancreatitis may develop later during the course of observation in patients with acute biliary tract disease, when it has not been noted on admission. In patients with acute biliary tract disease, hyperenzymemia may be absent on admission to the hospital, but may develop some days later during the period of observation. The possibility of development of acute pancreatitis should ever be in mind in those patients whose convalescence from an "acute biliary attack" is not satisfactory. Enzyme determinations should be frequently repeated under these circumstances. In three patients in this series, acute pancreatitis appeared to develop under our observation when serum enzyme tests had been negative on admission.

Hyperbilirubinemia and Acute Pancreatitis.—Because of the anatomic relationship of the lower end of the choledochus and the pancreas, it would not be difficult to explain the occurrence of hyperbilirubinemia merely on the basis of swelling of the pancreatic head. Nevertheless, it had been our clinical impression that, when hyperbilirubinemia accompanied acute pancreatitis, concomitant biliary tract disease was usually present. For this reason we analyzed our cases of acute pancreatitis associated with hyperbilirubinemia. Seventeen patients in this series showed hyperbilirubinemia (10 with obvious icterus). Fourteen of these had proven biliary tract disease. Information concerning the status of the biliary tract was not available in the remaining three patients. At the time of this compilation we have not a single instance of the occurrence of hyperbilirubinemia in association with acute pancreatitis in which concomitant biliary tract disease has been proved to be absent. Perhaps one might conclude that the association of hyperbilirubinemia with acute pancreatitis should suggest the existence of concomitant disease in the biliary tract.

NECROSIS VERSUS EDEMA

It has been mentioned that it may be difficult in some instances to appraise correctly the actual degree of inflammation in the pancreas based upon early observation of the patient. In the analysis of this material, certain features should suggest and others indicate that actual pancreatic necrosis is present. These features will be mentioned briefly.

(1) *Sex.*—Necrosis was more common in the female sex (54 per cent of the females compared to 25 per cent of the males).

(2) *Etiologic Factor.*—Two-thirds of our cases with necrosis occurred among patients with biliary tract disease; but fifty per cent of patients with edema of the pancreas also had biliary tract disease. Thus, there is possibly a slight increase in the incidence of pancreatic necrosis compared to edema, in women with biliary tract disease.

(3) *Clinical Features.*—The more severe pain was encountered in necrotic cases. Shock, when present, should suggest necrosis. Skin changes: a blotchy cyanosis was recorded in two patients, both were instances of necrosis. Thrombophlebitis had been noted in two patients, both with necrosis. A definite tendency toward higher temperatures (greater than 100° F.) and toward greater acceleration of the pulse rate (more than 100) characterized the cases of necrosis, as compared with edema of the pancreas. The degree of leukocytosis was helpful in this differential diagnosis. There were seven instances of leukocytic counts above 15,000 per 100 cm. in eleven patients with necrosis; whereas, in the cases of edema, only four of twenty-one patients had elevations above 15,000 leukocytes per 100 cm.

(4) *Duration of the Attack.*—On rigid therapy, prolongation of the attack with maintenance of clinical and laboratory features suggesting active inflammation persisting beyond three or four days strongly favors the presence of some degree of necrosis.

(5) *Acute Diabetes.*—There were three instances of acute diabetes that developed during the course of acute pancreatitis in this series of patients. All three patients were examples of pancreatic necrosis.

(6) *Hyperenzymemia.*—It has been mentioned that there is no significant correlation between the severity of the inflammatory process and the degree of elevation of the serum enzyme values. Whether or not a failure of the serum enzyme concentrations to approximate the normal within 5-7 days after the onset of the attack suggest that some degree of pancreatic necrosis remains to be determined with further experience.

(7) *Hypocalcemia.*—Our experience with this series of cases supports the thesis of Edmondson and Berne,[5] that a decided drop in blood serum calcium after the third day of illness strongly supports the existence of pancreatic necrosis rather than simple edema.

(8) *Electrocardiographic Changes.*—In sixteen of nineteen patients in whom electrocardiographic tracings were available, abnormalities were recorded. These changes were, for the most part, non-specific and temporary. In few instances the cardiologist could not differentiate the changes from those seen in coronary occlusion. Electrocardiographic changes were encountered with equal frequency in patients with edema and with necrosis. It should be emphasized that, in the differential diagnosis of myocardial infarction and acute pancreatitis, the pancreatic serum enzyme tests have greater value than electrocardiographic tracings.

(9) *Complications.*—The occurrence of complications such as spreading peritonitis, abscess, cyst formation, and hematoma, is to be anticipated only in patients with severe inflammation associated with necrosis.

THERAPY

The results of therapy in this group of patients is outlined in *Table 7*. Three of the five patients operated upon in the emergency were seen in the early years of this study, prior to the institution of preliminary rigid medical therapy. Twenty- eight patients have been treated by the conservative regimen to be outlined. Although seven of those were

THERAPY, ACUTE PANCREATITIS, 33 CASES

	Cases	Death
Operation, emergency	5	1
Conservative therapy, necrosis 7, edema 21	28	0
Operation, before discharge	7	0
For biliary disease	6	0
For abscess	1	0

TABLE 7

classified as instances of necrosis, there have been no deaths on our service since initial non-operative conservative treatment has been instituted. Obviously this good fortune cannot continue indefinitely.

The objectives in management of acute pancreatitis should be (1) to remove the causative factor, whenever possible; (2) to combat shock, dehydration and electrolyte imbalances; (3) to relieve pain and thereby its accompaniment of vasoconstriction to the pancreas; and (4) to suppress external pancreatic function by avoiding hormonal and nervous stimulation. In the interest of the first objective, immediate operation was previously performed frequently in the treatment of acute pancreatitis. The usual operation was cholecystostomy and drainage of the lesser sac or retroperitoneal space. It was reasoned that decompression of the biliary tract would reduce the probability of further reflux of bile into the pancreatic ducts. However, the mortality following immediate operation was in excess of 50 per cent. Perhaps the added stress of an operative procedure precipitated the exhaustion phase of the alarm reaction. It must be pointed out that this high mortality occurred in the era prior to enzyme determinations and the diagnosis was made only at the time of operation and then upon a selected group of patients in whom the severity of the clinical picture compelled exploration. The occurrence of necrosis was undoubtedly higher in this group of patients. It has never been clear to us what the surgeon could hope to accomplish at surgery, in the absence of a complication, that would favorably influence the pathologic process and benefit the patient. It is now generally agreed that immediate operation is unwise, providing "acute abdominal emergencies" can be excluded. It is thought best to withhold operation during the acute stage of pancreatitis unless it becomes complicated by suppuration, severe hemorrhage, or spreading peritonitis. After convalescence from the acute attack, if a severe complication has not developed, a definitive biliary tract operation is required in many patients.

The principles of treatment used in our patients follow:

Treatment of Shock, Dehydration, and Electrolyte Imbalances.—Every effort should be made to overcome shock as quickly as possible by the administration of plasma and blood transfusions as required. "Nothing by mouth" is instituted to avoid stimulation of external pancreatic secretion and perpetuation of vomiting. Therefore, sufficient parenteral fluids and electrolytes are provided to maintain adequate renal function and electrolyte balance. Diabetes mellitus occasionally accompanies acute pancreatic necrosis, and caution must be exercised in the administration of glucose and insulin. Usually 10 units of regular insulin are given subcutaneously with each 50 grams of glucose intravenously. Hypertonic solutions are avoided; usually, a 5 per cent solution of glucose is given by slow infusion. Frequent blood sugar determinations may be required to properly control hyperglycemia and, more important, to avoid insulin shock and hypoglycemia. Hypoglycemic stimulation of the vagal mechanism of pancreatic secretion may prove deleterious.

Relief of Pain.—As soon as the diagnosis has been established, or the course of action in therapy settled, pain should be relieved. Because severe pain is frequently accompanied by vasoconstriction, which is harmful to the myocardium and may reduce pancreatic blood flow sufficiently to convert simple edema to necrosis, every effort should be made to abolish pain promptly. Morphine should not be used because of its tendency to induce spasm in the sphincter of Oddi; its administration is frequently followed by nausea and vomiting. Demerol,® in a dose of 50 mg., has been looked upon as the analgesic of choice because of its atropine-like action. Recent reports suggest that demerol® in large doses likewise induces sphincteric and duodenal spasm. In combination with papaverine (2 grains intravenously) or nitroglycerine (grain 1/50-1/150), it is probably preferred to morphine or codeine. Sedation with sodium phenobarbital or paraldehyde (intramuscularly) may be given as needed. Paravertebral block has not been very helpful in the control of pain in acute pancreatitis, but the results with epidural block (continuous or intermittent) in initial experiences are promising.

Relaxation of the sphincter of Oddi is desirable to prevent further reflux of bile into the pancreatic duct. Antispasmodic agents

employed to relax the sphincter of Oddi are either myogenic or neurogenic in mechanism of action. The most effective myogenic antispasmodic drugs are papaverine, nitroglycerine, and amyl nitrate. Banthine® (25-50 mg. intramuscularly), demerol® (50 mg. intramuscularly) or atropine (1 mg. intramuscularly) are the most useful neurogenic antispasmodic agents.

Measures to Avoid Hormonal Stimulation of the Pancreas.—Protein food products and acid chyme are the most important stimuli of pancreatic secretion by the hormonal mechanism (secretin and pancreozymin). Thus no food is permitted by mouth when acute pancreatitis is suspected, and a nasal tube is introduced into the stomach for continuous or intermittent Wangensteen suction. Care must be taken to maintain the tip of the tube proximal to the pylorus in order to effectively reduce the passage of acid gastric juice into the duodenum. Soluble or insoluble alkalies may be administered every one or two hours during the period of intermittent Wagensteen suction. If these measures are carried out meticulously, it is likely that the hormonal phase of pancreatic secretion may be suppressed.

Measures to Avoid Nervous Stimulation of the Pancreas.—The nervous mechanism of pancreatic secretion is mediated via the vagus nerve. Vagal activity may be depressed by such drugs as atropine (1/60 to 1/120 grain every four hours), tetraethylammonium (200 to 400 mg. intravenously, avoid hypotensive doses) or banthine® (50 to 100 mg.). Atropine has been employed in most of our patients. We have not used tetraethylammonium because of its hypotensive effect. The volume of gastric secretion may also be reduced by depression of the vagus nerve, and thus secondarily suppress stimulation of the pancreas. Obviously, vagomimetic drugs, such as prostigmine or urecholine, which might conceivably be used to overcome abdominal distention, are to be withheld.

Other Measures.—Blood calcium determinations should be carried out, at least after the third day, and calcium gluconate administered parenterally if a calcium deficit is demonstrated. The effect of antibiotic agents or sulfonamides in the prevention of suppurative complications is difficult to evaluate, but their use prophylactically would appear justified.

The length of time in which the more rigid type of therapy is to be carried out depends upon the clinical status of the patient. Recovery from shock, return of the temperature to normal, a drop in the leukocyte count, and recedence of the physical evidences of inflammation constitute the most important guides. These patients must be watched carefully day by day to make sure that a complication is not developing which requires surgical intervention. A small percentage of patients will have a hematoma, an acute cyst, a large suppurating mass or spreading peritonitis, and of course, will have to be operated upon. After subsidence of the acute attack, the biliary tract should be studied. If choledocholithiasis or gallbladder disease is present, operation on the biliary tract is advised.

Often these patients do not remain well after subsidence of the acute attack. Many patients who have had an attack of acute pancreatitis, experience recurrences of either acute or subacute pancreatitis. As a consequence of chronic relapsing pancreatitis, occasionally diabetes mellitus, steatorrhea or calcification in the pancreas may develop. Subsequent attacks may be provoked by marked indiscretions in eating and overeating, and commonly follow alcoholic excess. These patients should be advised never to use alcoholic beverages.

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EDITORIAL DEPARTMENT

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SOUTHERN MEDICAL ASSOCIATION
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SODIUM METABOLISM AND PREMENSTRUAL TENSION

The premenstrual syndrome of edematous weight gain and abdominal fullness followed by intramenstrual diuresis and relief of symptoms is well known. It has been generally realized that abnormal saline retention is basically responsible for this distressing entity, since the control and maintenance of the normal volume of extracellular fluid is fundamentally related to the metabolism of sodium and chloride. Recent investigations have thrown new light on physiologic mechanisms involved.

Regulation of sodium excretion from the body is under the influence of the hormones of the adrenal cortex.[1] The principle sodium retaining hormone, an 11-desoxycorticosteroid, is elaborated by the adrenal cortex upon stimulation by the corticotrophic hormone of the anterior pituitary. This steroid acts primarily upon the renal tubular epithelium, resulting in reabsorption of sodium, chloride, and water. Sodium excretion in the sweat may also be affected.[2]

The administration of corticotrophin or desoxycorticosterone acetate promotes retention of sodium chloride and water, and the extracellular fluid volume increases.[3,4] Clinical edema frequently ensues.

It has been shown that estrogen oxidation products have marked anterior pituitary stimulating properties. On the other hand, intact estrogens and unoxidized estrogen products inhibit pituitary activity.[5] Low levels of progesterone, such as occur premenstrually, apparently promote the oxidation of estrogens and presumably, therefore, hypophyseal stimulation.[6] Such stimulation is manifested by increased amounts of follicle-stimulating hormone, luteinizing hormone, and prolactin. A rapid rise in the urinary concentration of these products occurs prior to the onset of menses. In addition, depression of serum diastase, present at this phase, is believed to reflect an increase in adrenal cortical activity secondary to corticotrophin release.[7] Similarly, it is at this time that an antidiuretic substance has been shown to be active, suggesting an increase in posterior pituitary activity as well.[8]

Increased systemic levels of corticotrophin promote the release of salt-retaining hormones by the adrenal cortex. This, in turn, causes excessive retention of salt by the renal tubular epithelium, and, to some extent, by the sweat glands.[3,4]

It cannot be overlooked that parenteral administration of large quantities of estrogen or progesterone will promote temporary sodium retention by the renal apparatus. However, it is believed that premenstrual fluid and electrolyte retention is not due to these agents, since their systemic concentration appears too low

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at this stage of the cycle to cause the amount of sodium retention usually seen.[8]

The evidence presented suggests that premenstrual sodium and water retention is a result of increased adrenal cortical activity. The release of salt-retaining hormones by the adrenal cortex is effected by the anterior pituitary, under the influence of estrogen oxidation products.

PROTEIN BOUND IODINE

The diagnosis of thyrotoxicosis may greatly perplex the conscientious physician, since the clinical picture of hyperthyroidism occasionally varies considerably from its classical description. Inasmuch as modern therapy of hyperthyroidism effectively affords relief from the damaging effects of the disease of myocardium, liver and other organs, the physician has a particularly great responsibility in establishing the diagnosis early and unequivocally. Heretofore, estimation of the basal metabolic rate has been the chief method of confirming clinical suspicion of the disease. Unfortunately, the basal metabolic determination may fail to provide help in those cases in which confirmation is most urgently needed. Exemplifying such cases are the persons suffering with chronic anxiety, certain patients with congestive heart failure, and the host of individuals who develop apprehension when confronted with a strange apparatus into which they are expected to breathe calmly and quietly. Furthermore, it has long been recognized that measurement of oxygen consumption *per se* is a rather indirect index of thyroid activity.

The recent introduction of the protein bound iodine (PBI) determination into clinical use as a more direct assay of thyroid gland function has met with approving though cautious response among physicians. Granted that the determination is subject to a number of technical pitfalls, and that the entity to be measured is of such infinitesimal magnitude that it represents one twenty-thousandth that of the blood sugar concentration (4-8 micrograms per cent of iodine compared with 80,000 to 100,000 micrograms per cent of blood sugar), this determination obviates many of the encumbrances and inaccuracies of the basal metabolic determination. Although it is hoped that increasing use of PBI determinations will reduce the incidence of technical errors, other precautions should be observed by those interpreting PBI data. It is recognized that the protein bound iodine may be falsely elevated following x-ray procedures employing iodine-containing media. The duration of such an elevation varies greatly with the procedure and with the rapidity of excretion of the dye. Although elevation of PBI resulting from intravenous pyelography may last for a number of days, and that caused by the dyes used in bronchography and cholecystography may last for many months,[1] residual dye from a myelogram may cause elevation of the PBI for years.[2] Physiological elevations of the PBI also occur. Pregnancy is normally accompanied by a rise in PBI by the sixteenth week; there is even a possibility that failure of the PBI to rise by that time may foretell an increased susceptibility to spontaneous abortion.[3] On the other hand, the PBI may be lowered by administration of mercurial diuretics.[4] Low values may also be found in the nephrotic syndrome, advanced cirrhosis, and in diabetes associated with intercapillary glomerulosclerosis.[5] Although inorganic iodine administration to hyperthyroid individuals may result in a fall in the PBI, a transient rise in PBI may follow administration of iodides to euthyroid individuals.[6]

The usefulness of the PBI determination is not confined to the diagnosis of thyrotoxicosis, myxedema or incipient abortion. Use of the PBI determination may be of particular value in evaluating non-hyperthyroid states associated with an increased basal metabolic rate, such as leukemia, chronic infections with fever, pulmonary tuberculosis, polycythemia vera, pheochromocytoma and Hodgkin's disease. Serial determinations of PBI are of value in evaluating the effectiveness of antithyroid therapy, whether mediated by surgical or med-

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ical means. Studied judiciously, with full awareness of the clinical findings, and interpreted in the light of all possible interfering factors, the PBI determination offers great promise as a diagnostic aid.

TWENTY-FIVE YEARS AGO

From Journals of 1928

Races and Basal Metabolism.[1]—In connection with an archaeological expedition to Chichen Itza, Yucatan, the basal metabolism of white men and women and of Maya Indians was studied. Measurements made on white members of the expedition before, during and following a short stay in Yucatan and on other white men and women who had been in Yucatan less than four months, indicated that the change in environment was without influence upon the basal metabolism, thus emphasizing the absence of an effect of the sub-tropical climate. A group of thirty-two Mayas (male) were found to have a metabolism averaging 5.2 per cent above the standards for white men of similar age, weight and height living in the northern part of the United States. . . . . Since the sub-tropical climate might be expected to lower rather than raise the metabolism of these Mayas, it is evident that some other factor, presumably racial, has asserted itself. The contrast between the present-day civilization of Orientals, whose basal metabolism has been shown to be low, and of these Mayas argues against interpreting a low metabolism as an index of racial inferiority or a high metabolism as an index of racial superiority.

Effect of Ultra Violet and Other Light upon Parathyroid Glands of Chicks.[2]—Hyperplasia of the parathyroid glands is a frequent observation among students of those diseases involving a disturbance of the normal calcium and phosphorus metabolism. Erdheim . . . noted marked hyperplasia of the parathyroids of animals with spontaneous rickets. Pappenheimer and Minor . . . in . . these glands in cases of rickets in human beings recognized marked increase in the number of cells. Luce (1923) concluded that the calcium content of the diet bears a direct relationship to the size of the gland. In rats maintained on a diet low in calcium for twelve weeks, the parathyroids were many times larger than normal. . . . . .

Pens were constructed and screened on their southern exposures by amber, blue and ordinary glass, and vitaglass, thus transmitting variable portions of the sun's spectrum . . . . . vitaglass permits the maximal transmission of ultraviolet irradiation . . . . to 272 millimicrons. . . . . Enlargement of the parathyroid glands was recognized within the first few weeks in chicks growing under amber and blue glass; this enlargement was due to hyperplasia rather than hypertrophy . . . . Regression of the cell columns is manifested early; . . . . . Cysts accompany retrogressive processes . . These cysts are of two types. One is walled off from the remaining portion of the gland by definite columnar epithelium while the other is enclosed by a series of concentrically arranged flattened cells.

There was no organic indication either in blood calcium, blood phosphorus or general activity that such parathyroid hyperplasia had arisen.

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AN HISTORIC EVENT IN AMERICAN MEDICINE

The House of Delegates of the American Medical Association met in called session at the Statler Hotel, Washington, D. C., Saturday, March 14, at 10:00 a.m. There were one hundred and seventy-nine delegates present out of a possible one hundred and eighty-three. Only two or three times before in the more than one hundred years of the existence of the American Medical Association has there been a special called meeting of the House of Delegates.

The session was presided over by the Speaker of the House, Dr. James R. Reuling, Bayside, New York.

The invocation was given by the Rev. Edward L. R. Elson, D.D., Pastor of the National Presbyterian Church, Washington, the Church of which President and Mrs. Eisenhower are members.

The President, Dwight D. Eisenhower, addressed the House on the policy of the Administration particularly as to Reorganization Plan No. 1 for a Department of Health, Education and Welfare. The President was accompanied by Mrs. Oveta Culp Hobby, now Administrator of Federal Security Agency, who will be advanced to cabinet rank if Reorganization Plan No. 1 is adopted. After the President had spoken and left, the House was addressed by Honorable Robert A. Taft, Senator from Ohio, and Honorable Walter H. Judd, a doctor of medicine, Congressman from the Fifth District of Minnesota.

The President of the American Medical Association, Dr. Louis H. Bauer, New York, addressed the House. Dr. John W. Cline, San Francisco, California, Immediate Past President of the American Medical Association, in most appropriate remarks, presented Dr. Elmer L. Henderson, Louisville, Kentucky, Past President of the American Medical Associtation and Chairman of its former Coordinating Committee, and Chairman of the Professional Committee for Eisenhower and Nixon during the campaign last year, who addressed the House. Dr. Henderson is a Past President of the Southern Medical Association and now Chairman of its Board of Trustees.

The Chairman of the Board of Trustees of the American Medical Association, Dr. Dwight H. Murray, Napa, California, presented the following report for the Trustees:

> "The House of Delegates of the American Medical Association has for nearly 80 years been on record as favoring an independent De-

partment of Health in the federal government. The reason for this stand has been that the House has felt that health and medicine should be given a status commensurate with their dignity and importance in the lives of the American people, and that they should be completely divorced from any political considerations.

"The Board of Trustees, after a careful study of the policy of the American Medical Association with respect to the administration of health activities in the Executive Branch of the government and after studying the Reorganization Plan for elevation of the Federal Security Agency to cabinet status submitted by President Eisenhower to the Congress, finds that Reorganization Plan No. 1 of 1953 provides for a special assistant to the Secretary for Health and Medical Affairs. This provision is a step in the right direction which should result in centralized coordination under a leader in the medical field of the health activities of the proposed department. Health, therefore, is given a special position. The proposed plan, properly administered, will permit more effective coordination and administration of the health activities of the new Department without interference or control by other branches.

"Previous attempts to raise the Federal Security Agency from an independent agency to the level of an Executive Department have been opposed by the Association because the plan did not meet these aims.

"Inasmuch as federal health benefits and programs are established by the Congress, an administration bent on achieving the nationalization of medicine cannot reach that goal except with the support of Congress. Therefore, an organizational plan through which federal health activities are administered, although important, is not nearly so vital an issue as the policies adopted by the Congress of the United States.

"The Board of Trustees recommends that the House of Delegates reaffirm its stand in favor of an independent Department of Health but that it support the Reorganization Plan No. 1 of 1953 as being a step in the right direction; that the American Medical Association cooperate in making the plan successful and that it watch its development with great care and interest.

"It should be understood, however, that the Association reserves the right to make recommendations for amendment of the then existing law or to press for the establishment of an independent Department of Health, if the present plan does not, after a sufficient length of time for development, result in proper advancement in and protection of health and medical science and in their freedom from political control."

The Report of the Board of Trustees was approved by a large majority vote and then made unanimous.

This is the first time in the history of the American Medical Association that a President of the United States has visited its House of Delegates and spoken to it. An appropriate resolution of thanks and appreciation to President Eisenhower was passed unanimously after he had left the room.

While Reorganization Plan No. 1 does not meet the overall wishes of organized medicine in the United States it was the consensus of a great majority of those present that it was a movement in the right direction and gave medicine a better place in the federal government than it has previously had in any administration in Washington. It was very obvious from the address of the President that he is friendly to the medical profession. Those speaking to the House who had conferred with Mrs. Hobby, who will be Secretary of the Department of Health, Education and Welfare, indicated that she too is friendly to the medical profession and will appoint a doctor of medicine as special assistant in charge of health and medical affairs.

Truly this meeting was an historic event in medicine in the United States.

Correspondence

Shreveport, Louisiana, February 24, 1953

Editor, SOUTHERN MEDICAL JOURNAL:

Referring to the correspondence in the February number, between Admiral Boone and Dr. Wilkinson, I wish to state that I heard Dr. Wilkinson's address and felt (and still feel) that it was very timely. I have had the pleasure of meeting Admiral Boone and know him to be an excellent gentleman, one of unimpeachable character, of best intentions, but I cannot get it out of my mind that, although the Admiral is Chief Medical Director of the Veterans Administration, still he does not know all the inside workings and conditions of the various hospitals. Ever since I learned that the local hospital staff is in active competition with private practice, on direct orders from headquarters, in that insured accident cases are, under these orders, referred to the Veterans Administration Hospital instead of to private institutions, I have resigned my connection with the local hospital. In addition, I could cite cases where patients have been hospitalized for non-service connected disabilities and remained for many months in the Veterans Administration Hospital when they had Hospital Association benefits, by which they could have gone to a private hospital, and not been treated at the expense of the Veterans Administration, which means all of us.

This letter is not intended as an affront to Admiral Boone or to others connected with the management of the Veterans Administration, but I feel that if Dr. Wilkinson's statements are left unsupported, it will have a tendency in part to suppress free speech.

(Signed) A. A. HEROLD, M.D.

Book Reviews

Research in Endocrinology. By August A. Werner, M.D., Assistant Professor of Internal Medicine, St. Louis University School of Medicine, St. Louis, Missouri, and Associates. Edited by Al R. Schmidt, City Editor, Belleville Daily Advocate, Belleville, Illinois. 285 pages with illustrations. St. Louis: Dr. August A. Werner (403 Humboldt Building), 1952.

This interesting little volume is in general an autobiography of Dr. Werner including a collection of his endocrine research projects. The book contains a great deal about his personal life and thoughts and a bibliography of his past publications and exhibits. Many of his reprints are published completely as part of this book.

Most of his work has been on the climacteric syndrome and the effect of estrogenic hormones as a treatment of this difficulty. A great deal has also been written upon the male climacteric and on involutional melancholia.

It is of interest and value to friends of Dr. Werner.

Practical Dermatology. For Medical Students and General Practitioners. By George M. Lewis, M.D., F.A.C.P., Professor of Clinical Medicine (Dermatology), Cornell University Medical College. 328 pages with illustrations. Philadelphia: W. B. Saunders Company, 1952. Price $7.50.

The book is designed as a text for medical students, and a guide for general practitioners. Well illustrated, it describes the more common skin disorders, with main emphasis on diagnosis.

Discussions of basic fields which pad most texts are omitted.

The beautiful format, the division of the material into symptoms, differential diagnosis, etiology and treatment, with important portions in heavy print, make it particularly easy to find desired information. The 99 plates with an average of four pictures, clear and lucid, to the plate make visual diagnosis possible.

New forms of treatment, especially cortisone and ACTH therapy, are covered both in the text, and in the excellent dermatologic formulary at the end of the book. Contentious and confusing material has been omitted.

The Esophagus and Its Diseases. By Eddy D. Palmer, M.D., F.A.C.P., Lieutenant Colonel, Medical Corps, United States Army; Chief, Gastrointestinal Section, Walter Reed Army Hospital. 553 pages with illustrations by Phyllis Anderson, some in color. New York: Paul B. Hoeber, Inc., 1952. Price $15.00.

This book is 553 pages long and has some beautiful x-rays of the common esophageal diseases. Some criticism is in order.

The therapy recommended for achalasia and cardiospasm is outmoded, since the Heller operation is not described as the operative procedure of choice. No mention is made of urging the patients to sleep with the head of the bed elevated (to prevent regurgitation of the esophageal contents into the tracheobronchial tree with the resulting aspiration pneumonitis).

The chapter on congenital atresia does not emphasize the importance of not giving barium as a contrast medium. Bariums may cause a severe pneumonitis.

The discussion of cancer of the esophagus does not mention the necessity of bronchoscoping the patient to determine whether there has been any extension of the tumor into the tracheobronchial tree.

The chapter on diaphragmatic hernia does not emphasize the masquerading effects of the condition, and the indications for operation are not clear. Conservative management fails to mention the most important thing, which is having the patient sleep with the head of the bed elevated.

The author reports that acquired shortening of the esophagus is much less common than has previously been supposed, although actually, a congenital short esophagus is very rare. Most cases of short esophagus are acquired and are a complication of a diaphragmatic hernia of long standing.

The author's treatment of stricture of the esophagus is daily dilation of the stricture with multiple esophagoscopic examination. This is dangerous and unnecessary. Too frequent dilation can cause as much stricture as the original corrosives. Only a patient in the Army or a charity institution could afford the treatment outlined.

There are excellent chapters on clinical physiology, diverticulosis, varices and hemorrhage, and diffuse collagen diseases.

Pardon My Sneeze. By Milton Millman, M.D., Fellow American Academy of Allergists, Member American Academy of Allergy. 215 pages with illustrations. San Diego: Frye & Smith Ltd., 1952.

Dr. Millman's book is intended as guidance and instruction for allergic patients. Much of it is written as questions and answers. The questions have been asked of the author by his patients and his answers are given.

There is a general discussion of allergy followed by discussions of the separate syndromes, asthma, hay fever, eczema and the others. Enough is given on methods of diagnosis so that the patient can understand what is being attempted.

Sources of contact with the miscellaneous inhalant allergens, house dust and the others, are given and methods of avoidance. Food allergy is discussed extensively and sources of contact with the major food allergens milk, egg, wheat and corn are considered.

Finally there are several chapters containing numerous recipes for preparation of diets free of the major food allergens milk, egg, wheat and corn. Many patients will find this the most helpful part of the book.

The illustrations are misleading since by them one is caused to expect the material to be handled in

lighter vein, which is not the case. An author should list the societies in which he claims membership by their correct names. There is an American College of Allergists and an American Academy of Allergy. By his listing one is left in doubt whether he claims membership in one or both.

There is little in this work to criticize and much to praise.

The History of American Epidemiology. By C. E. A. Winslow, Dr.P.H., Professor Emeritus, Yale University School of Medicine; Editor, American Journal of Public Health; Wilson G. Smillie, M.D., Professor and Chairman, Department of Public Health and Preventive Medicine, Cornell University Medical College; James A. Doull, M.D., Medical Director, Leonard Wood Memorial (American Leprosy Foundation); John E. Gordon, M.D., Professor and Chairman, Department of Epidemiology, School of Public Health, Harvard University. Edited by Franklin H. Top, M.D., Professor of Epidemiology and Pediatrics, College of Medical Sciences, University of Minnesota. 190 pages. St. Louis: The C. V. Mosby Company, 1952. Price $4.75.

The four papers of this monograph were originally presented at the Twentieth Anniversary Session of the Epidemiology Section of the American Public Health Association. The authors trace the evolution of epidemiology in America through four periods: the colonial era; the period of great epidemics (1800-1875); the bacteriological era (1876-1920); and the recent past and present, with some predictions for the foreseeable future. It is fascinating to follow the tremendous changes which have occurred in the problem of mass disease since the arrival of the pilgrims at Plymouth, and to review the impressive contributions made by American workers in such fields as yellow fever, rickettsial diseases, and tularemia, to name a few. The first essay commands attention not only for medical reasons, but also because it sheds some light upon the cultural background of the colonial period and some of its leaders: Franklin, Benjamin Rush, Noah Webster. As to the future, it is believed that epidemiology will find application in mass health problems other than those of communicable disease: for instance, in nutritional disorders, cancer, and even emotional and mental disorders.

Personal and Community Health. By C. E. Turner, A.M., Ed.M., D.Sc., Dr.P.H., Professor of Public Health Emeritus, Massachusetts Institute of Technology. Ninth Edition. 659 pages with illustrations. St. Louis: The C. V. Mosby Company, 1952. Price $4.25.

This is a comprehensive textbook on personal hygiene and public health problems, written primarily for instruction at the college level. The first part of the book is devoted to such aspects of personal health as nutrition, digestion, hygiene of the organs of respiration, circulation, and excretion, the nervous system, mental health, dermatologic, orthopedic and sex hygiene. While fundamentals of anatomy and physiology are considered, the greater part of each chapter contains good, common sense advice on health matters for the layman. The author, an experienced teacher, addresses himself mainly to young people and presents evidence in a scientific and detached manner, without moralizing, in discussing matters like alcohol and drug addiction. He stresses health maintenance which is "relatively simple, and immensely rewarding" for the well informed. Self-medication is, in general, discouraged. The second part of the book deals with some basic aspects of public health: communicable diseases, maternal and child health, sanitation, school and industrial health, and other community problems. In an appendix, a portion of the report on communicable diseases of the American Public Health Association has been reprinted. While this book is of value primarily to college students and nurses, any intelligent layman should find it instructive and rewarding.

Essentials of Histology. By Margaret M. Hoskins, Ph.D., and Gerrit Bevelander, Ph.D., New York University, New York City. Second Edition. 240 pages with 135 text illustrations and 2 color plates. St. Louis: The C. V. Mosby Company, 1952. Price $4.00.

In this, the second edition, the presentation of the material has not been changed from the original nor the arrangement of the chapters, but several sections have been revised or rewritten with new material added in a number of instances. The authors have attempted to correlate the physiology and biochemical functions along with the histology of tissue. The whole content is well organized so that the essential parts may be digested with a minimum of reading.

Rypins' Medical Licensure Examinations. Topical Summaries and Questions. Edited by Walter L. Bierring, M.D., F.A.C.P., M.R.C.P., Edin. (Hon.), Former Member, National Board of Medical Examiners, American Board of Internal Medicine, Iowa State Board of Medical Examiners; Professor Emeritus, Theory and Practice of Medicine; College of Medicine, State University of Iowa. With the collaboration of a Review Panel. Seventh Edition. 856 pages. Philadelphia: J. B. Lippincott Company, 1952. Price $8.00.

"Rypins" has become a trusted stand-by for medical graduates in preparing for licensing examinations. The original author said: "It is taken for granted that the student has been adequately trained in the medical sciences and there is no attempt to teach him anything new." The purpose of the book is, rather, to review in an orderly manner the huge amount of information acquired during the medical course. This is accomplished by means of summaries in thirteen major fields, covering both the basic medical sciences and the clinical sciences. A selection of typical examination questions at the end of each section helps the reader to test his knowledge.

In this new edition, the chapters on biochemistry (formerly chemistry), microbiology (formerly bacteriology), and preventive medicine and public health (formerly hygiene and preventive medicine) have been

completely rewritten. The narrative and the questions in the other chapters have also been revised by the editor and a review panel. Study of this book prior to examination will help the candidate to refresh his memory and to rearrange his material. A systematic review, and the assurance that he is able to answer many actual examination questions, will also tend to allay his fears of the approaching ordeal.

Viral and Rickettsial Infections of Man. Edited by Thomas M. Rivers, M.D., Director of the Hospital, The Rockefeller Institute for Medical Research. Second Edition. 719 pages with 90 illustrations, including 7 plates in color. Philadelphia: J. B. Lippincott Company, 1952. Price $7.50.

Not many years ago, the study of viral and rickettsial diseases in medical schools occupied but a small portion of the bacteriology curriculum. Within the past ten to fifteen years, knowledge in the field of microbiology has greatly expanded, largely in the realm of these borderline microscopic and submicroscopic organisms. Bacterial diseases no longer constitute such a grave clinical problem as in former years. Even rickettsiae are being conquered by the new antibiotics, but most viral diseases continue to run a clinical course unmodified by current therapy. Fortunately, progress is constantly being made in the study of viruses, and with the publication of their second edition, Dr. Rivers and his colleagues, long noted for their studies on this subject, have added much new material to an already comprehensive text.

New chapters have been added on hemagglutination by viruses, and interference between animal viruses. There is a particularly informative chapter on coxsackie viruses, as well as another of great interest to clinicians dealing with the diagnostic aspects of virus and rickettsial diseases.

1951 Annual Report of the John and Mary R. Markle Foundation. George Whitney, President. 83 pages. New York: The John and Mary R. Markle Foundation, 1952.

The above-named foundation has supported various charitable, scientific, and educational enterprises during a period of 25 years. At the present time, it provides grants to "Scholars in Medical Science," who are faculty members on the staffs of 49 medical schools. This report sketches the history of the Foundation and the work it has performed. It furthermore includes a list of new grants, a description of scientific projects supported by the Foundation, and a financial report.

Books Received

Fundamentals of Clinical Cancer. With Emphasis on Early Diagnosis and Treatment. By Leonard B. Goldman, M.D., Clinical Professor of Radiotherapy, New York Medical College, Flower and Fifth Avenue Hospitals, New York, New York. 312 pages with illustrations. New York: Grune and Stratton, Inc., 1953. Price $8.75.

Fundamentals of Clinical Orthopedics. By Peter A. Casagrande, M.D., Orthopedic Department of Buffalo General Hospital, Buffalo Children's Hospital, and the University of Buffalo Medical School, Buffalo, New York; and Harold M. Frost, Jr., M.D., Orthopedic Department of Buffalo General Hospital and the University of Buffalo Medical School, Buffalo, New York. 582 pages with illlustrations. New York: Grune and Stratton, Inc., 1953. Price $18.50.

Familial Nonreaginic Food—Allergy. By Arthur F. Coca, M.D., Oradell, New Jersey. Third Edition. 279 pages. Springfield, Illinois: Charles C. Thomas, Publisher, 1953. Price $10.50.

The Official Preparations of Pharmacy. By Charles Oren Lee, Ph.D., Professor of Pharmacy, Purdue University School of Pharmacy, Lafayette, Indiana. Second Edition. 544 pages with illustrations. St. Louis: The C. V. Mosby Company, 1953. Price $5.50.

Liver Injury. By F. W. Hoffbauer, M.D., Associate Professor, Department of Medicine, University of Minnesota Hospitals, Minneapolis, Minnesota. First Edition. 265 pages with illustrations. New York: Josiah Macy, Jr. Foundation, 1952. Price $4.00.

The Principles of Neurological Surgery. By Loyal Davis, M.S., M.D., Ph.D., D.Sc., (Hon.) Professor of Surgery and Chairman of the Division of Surgery, Northwestern University Medical School, Chicago, Illinois. Fourth Edition. 544 pages with illustrations. Philadelphia: Lea & Febiger, 1953.

Poliomyelitis. By W. Ritchie Russell, C.B.E., M.D. (Edin.), M.A. (Oxon), F.R.C.P. (Edin.), F.R.C.P. (Lond.), Consultant Neurologist to the United Oxford Hospitals. 84 pages with illustrations. Baltimore: The Williams & Wilkins Company. Price $3.00.

Pediatric Nursing. By Gladys S. Benz, R.N., B.S., M.A., Assistant Supervisor and Instructor, Pediatric Nursing, Bellevue Schools of Nursing, New York. 624 pages with 122 illustrations. Second Edition with Teachers' Guide. St. Louis: The C. V. Mosby Company, 1953. Price $5.25.

Bedside Diagnosis. By Charles Seward, M.D., F.R.C.P. (Edin.) Honorary Physician, Royal Devon and Exeter Hospital; Consulting Physician, Princess Elizabeth Orthopoedic Hospital, West of England Eye Infirmary and The Ministry of Pensions: Honeyman Gillespie Lecturer: Late Adviser in Medicine to Eastern Command, India; Deputy President, Review Medical Board, India. Second Edition. 380 pages. Baltimore: The Williams & Wilkins Company, 1952. Price $3.50.

Symptoms and Signs in Clinical Medicine: An Introduction to Medical Diagnosis. By E. Noble Chamberlain, M.D., M.Sc., F.R.C.P., Senior Lecturer in Medicine, University of Liverpool; Physician in Charge, Medical Unit, Sefton General Hospital, Liverpool. Fifth Edition. 480 pages with illustrations. Baltimore: The Williams & Wilkins Company, 1952. Price $8.00.

Textbook of Virology. For Students and Practitioners of Medicine. By A. J. Rhodes, M.D., F.R.C.P. (Edin.), Research Associate, Connaught Medical Research Laboratories, and Professor of Virus Infections, School of Hygiene, University of Toronto: Virologist, Hospital for Sick Children, Toronto; and C. E. van Rooyen, M.D., D.Sc. (Edin.), M.R.C.P. (Lond.), Research Member, Connaught Medical Research Laboratories, and Professor of Virus Infections, School of Hygiene, University of Toronto. Second Edition. 561 pages with illustrations. Baltimore: The Williams and Wilkins Company, 1953. Price $8.00.

BCG Vaccination: Studies by the WHO Tuberculosis Research Office, Copenhagen. World Health Organization Monograph Series Number 12. Report prepared under the direction of Lydia B. Edwards, M.D., Chief of Field Studies; and Carroll E. Palmer, M.D., Ph.D., Director; with the assistance of Knut Magnus, cand. act., Assistant Statistician, 307 pages. Geneva, Switzerland: World Health Organization, 1953. Price $3.00. United States sales agent: Columbia University Press, International Documents Service, 2960 Broadway, New York 27, New York.

Textbook of Pediatrics. By Fanconi and Wallgren. Edited by W.R.F. Collis, M.A., M.D., F.R.C.P., F.R.C.P.I., D.P.H., Lecturer in Paediatrics, Dublin University; Physician, National Children's Hospital; Director of Department of Paediatrics, Rotunda Hospital, Dublin. 1,104 pages with illustrations. New York: Grune & Stratton, 1952. Price $19.50.

An Atlas of Skull Roentgenograms. By Bernard S. Epstein, M.D., Associate Radiologist, The Jewish Hospital of Brooklyn, Brooklyn, New York; and Leo M. Davidoff, M.D., Neurosurgeon, Mount Sinai Hospital, New York, and Director of Neurological Surgey, Beth Israel Hospital, New York, New York. 415 pages with 603 illustrations. Philadelphia: Lea and Febiger. 1953. Price $15.00.

Southern Medical News

ALABAMA

Dr. Frank L. Chenault, Decatur, will be Alabama's guest of honor at the First Western Hemisphere Conference of the World Medical Association, Richmond, Virginia, April 23-25, held in observance of the lengthening of life and the constant improvement of human health.

Dr. A. E. Casey, pathologist at the Birmingham Baptist Hospital, Birmingham, has been engaged in cancer research for a number of years, the research being financed by the hospital and Dr. Casey personally. He and his staff for the past two summers have spent several months at Jackson Memorial Laboratory, Bar Harbor, Maine, carrying on various phases of the work. Dr. Casey spoke on his work in cancer research at Baylor University College of Medicine, Houston, Texas, in February.

ARKANSAS

University of Arkansas School of Medicine, Little Rock, has received a $7,000 grant from the Arkansas Heart Association for study of the relation between chronic heart disease and disorders of the endocrine glands under the supervision of Dr. Benjamin B. Wells, professor of medicine. Drs. James T. Wortham, Paul Starr, Owen W. Beard, James E. Doherty and O. Boyd Houchin, Ph.D., will also be participating in the study.

Dr. A. C. Curtis has resigned from the Arkansas State Board of Health to enter service with the United States Public Health Service.

Dr. Wayne Stone has joined the staff of the DeQueen Clinic.

Dr. W. C. Hays has been elected a director of the Marianna-Lee County Chamber of Commerce.

Dr. F. F. Ferguson, Nashville, and Dr. Gordon P. Oates, Little Rock, have been recalled to active duty with the Naval Medical Corps.

Dr. W. A. Bullock, formerly of Elk City, Oklahoma, has taken over the practice of Dr. W. J. B. Williams at Cotton Plant. Dr. Williams has moved to Henning, Texas.

Dr. and Mrs. J. W. Morris, McCrory, recently celebrated their fifty-first wedding anniversary.

Dr. Wayne Stanfield has joined Dr. T. E. Williams and Dr. H. M. Baird, Newport, for the practice of eye, ear, nose and throat.

Dr. D. C. Lee and Dr. W. R. Lee have been installed as coroner and deputy coroner, respectively, of Garland County.

Dr. James W. Peavey has moved from Pine Bluff to Monticello.

Dr. Oscar Gray, Jacksonville, and Dr. Ralph G. Kramer, Fort Smith, have been recalled to active duty with the Naval Medical Corps.

Dr. Wayne Biggs, Clinton, has entered active duty with the Army Medical Corps.

DISTRICT OF COLUMBIA

Dr. Arthur Carlisle Christie, Washington, was honored on December 20 in recognition of his fifty years of distinguished service to his profession and to the community when a dinner was given by his friends in the medical profession and the Rotary Club of Washington at the Shoreham Hotel. Some two hundred prominent physicians and laymen paid tribute to Dr. Christie on this occasion, a number from distant places. Dr. and Mrs. Christie now reside at Crescent City, Florida.

National Conference on World Health will be held at Washington, Shoreham Hotel, convening in the evening of April 5 and adjourning at 3:00 p.m. April 7. This is a citizens' conference to discuss problems involved in world health work and facing the World Health Organization.

Dr. Andrew J. Marchetti, Washington, is Chairman of the Program Committee for the second clinical session of the American Academy of Obstetrics and Gynecology which will be held in Cincinnati, Ohio, Netherland Plaza Hotel, December 14-16 under the presidency of Dr. Robert A. Kimbrough, Jr., Philadelphia, Pennsylvania. Dr. Bayard Carter, Durham, North Carolina, is president-elect.

The office of the American Medical Association's Committee on Blood has been transferred from Washington to AMA headquarters in Chicago, and Dr. Paul L. Wermer, secretary of the AMA Committee on Research, succeeds Dr. Frank E. Wilson as secretary of the Committee on Blood.

Children's Hospital, Washington, a new $3,300,000 structure, was opened officially in December. This hospital, founded in 1870, has grown from four physicians to a present consulting staff of 19 physicians, an attending staff of 45 physicians, associate and junior associate staff of 183 physicians and a resident and extern staff of 25 physicians, these including representatives from every region in the United States. The building includes an auditorium named in honor of the late Dr. Joseph S. Wall, until recently Chief of the Medical Staff. Dr. Edgar P. Copeland is Chief of the Medical Staff, having come to the hospital in 1898 as a resident student, and Dr. Montgomery Blair, Jr., is Medical Director.

Dr. Henry L. Darner has been elected chairman of the Medical Advisory Board of the Planned Parenthood Association of the District of Columbia to succeed Dr. Walter Stokes, resigned. New members of the board are Drs. Henry D. Ecker, Maurice H. Friedman, Caroline Jackson, Shirley S. Martin, Neel J. Price, William T. Spence and Marshall deG. Ruffin; and physicians to serve on the Executive Committee are Drs. Isadore M. Alpher, Albert S. Bright, George Nordlinger, Carolyn Pincock, and Maurice A. Selinger.

Dr. Monroe J. Romansky, associate professor of medicine, George Washington University School of Medicine, Washington, as consultant in internal medicine to the Surgeon General of the Army recently made a six-weeks' tour of Army hospitals and installations in Germany and Austria at which time he addressed the Royal Society of Medicine in London, the Geneva Medical Society in Switzerland, and the Pasteur Institute in Paris on current trends in antibiotic therapy.

Dr. Winfred Overholser, Washington, was the official representative of the American Psychiatric Association at the International Congress of Neuropsychiatry held in Santiago, Chile, in December. He was one of three delegates who were made honorary members of the Medical Faculty of the University of Chile.

Dr. Donald Walters has been released from military service and has returned to his practice in Washington.

Dr. Arden Freer, Washington, recently received the Veterans Administration's highest award, the Exceptional Service Medal, the citation being the fourth such award to be presented by VA. Dr. Freer retired as Deputy Chief Medical Director of Veterans Administration in December.

Dr. Sydney Ross, Washington, presented a paper before the first International Antibiotic Congress held in Buenos Aires, Argentina, in December.

American Hearing Society, Washington, is sponsoring the twenty-fifth annual observance of National Hearing Week May 3-9. The organization is nonprofit. Affiliated with it are 115 local chapters in 34 states, the District of Columbia and Canada. It is supported by membership dues from individuals and chapters, and contributions from individuals and Community Chests. National Hearing Week is for the purpose of informing the public about existing programs for prevention of deafness, conservation of hearing and rehabilitation of the hard of hearing, and to point out the urgent need for extended services for children and adults whose hearing is below par. The theme for the campaign is "Hearing Is Priceless—Protect It." Membership in the Society is open to anyone interested in its program.

FLORIDA

A group of distinguished Cuban physicians presented a scientific program for the members of the Dade County Medical Association at Jackson Memorial Hospital, Miami, in January. Dr. Ralph W. Jack, president of DCMA, gave an address of welcome.

Dr. Andrew J. Leon, Miami Beach, has discontinued his private practice of medicine to go into active duty with the army.

Dr. Phillip R. Rezek, Miami, was invited by the staff of the Lago Oil & Transport Company in Aruba, and by the Medical Society in Curacao, Netherlands West Indies, to render a series of lectures in January covering "Pathology of Unexpected, Spontaneous Death," "Pathology of Pyelonephritis in Pregnancy, and Sequela," and "Conception and Limitations of Malignancy."

Bids were received in January for construction of a third floor to Doctors' Hospital, Coral Gables, increasing the bed capacity to 168 with an out-patient department. Dr. Arthur H. Weiland is chairman of the board; Dr. C. Howard McDevitt, Jr., vice-chairman; Dr. A. Daniel Amerise, treasurer; and Mrs. Mary J. Reeder, superintendent.

Dade County Pediatric and Orthopedic Societies will hold a joint seminar at the Jackson Memorial Hospital and Variety Children's Hospital, April 23-25.

Dr. Arthur J. Butt, Pensacola, has been elected to membership in the New York Academy of Sciences (Section of Physics and Chemistry).

Dr. Sullivan G. Bedell, Jacksonville, has been elected president of the Florida Association for Mental Health.

Dr. J. Brown Farrior, Tampa, recently presented a paper before the First Central American Congress of Otorhinolaryngology in San Salvador and at that time visited in Managua, Nicaragua and presented another paper in San Jose, Costa Rica.

Continued on page 52

8

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*Gordon, H. H.: Feeding of Premature Infants, Am. J. Dis. Child. June 1947.

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3. Temporal branch of facial nerve
4. External carotid artery & posterior facial vein
5. Superficial cervical lymph nodes
6. External jugular vein
7. Accessory nerve & internal carotid artery
8. Platysma muscle
9. Fourth cervical nerve
10. Superior position of sternocleidomastoid muscle
11. Deep cervical lymph nodes
12. Fifth cervical nerve
13. Posterior supraclavicular nerve & anterior jugular vein
14. Superficial cervical artery & vein
15. Middle supraclavicular nerve & subclavian artery
16. Transverse scapular artery & vein
17. Inferior position of sternocleidomastoid muscle
18. External maxillary artery & anterior facial vein
19. Submaxillary lymph nodes & digastric muscle
20. Submaxillary gland & mylohyoid muscle
21. Submental lymph nodes & hypoglossal nerve
22. Superior laryngeal artery & nerve
23. Superior cervical ganglion
24. Superior laryngeal vein & omohyoid muscle
25. Superior thyroid artery & vein
26. Ansa hypoglossi
27. Common carotid artery & sternothyroid muscle
28. Middle cervical ganglion & phrenic nerve
29. Vagus nerve
30. Thyroid gland & middle thyroid vein
31. Internal jugular vein
32. Sternohyoid muscle
33. Jugular lymphatic trunk
34. Inferior thyroid veins

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Continued from page 404

Dr. Homer L. Pearson, Jr., Miami, has been reappointed to a four-year term on the State Board of Medical Examiners.

Dr. Joseph C. Hayward, formerly of Orlando, has opened an office in San Diego, California, for the practice of urology.

GEORGIA

The Medical Association of Georgia will hold its 103d annual meeting in Savannah, DeSoto Hotel, May 10-13.

Georgia Heart Association will hold its fifth annual meeting in Savannah, DeSoto Hotel, September 4-5.

Houston-Rentz Clinic and Merritts Hospital in Colquitt have merged into the Southwest Georgia Hospital, Inc., with Dr. William Henry Houston, president; Dr. James W. Merritt, vice-president; Dr. Hinton J. Merritt, treasurer; and Dr. Turner W. Rentz, secretary.

Emanuel County Hospital has elected to its medical staff Dr. C. E. Powell, chief, succeeding Dr. D. D. Smith, who resigned to operate his own private hospital.

Warren Candler Hospital has elected Dr. Harold M. Smith, chief of staff; Dr. H. Y. Righton, vice-president of staff; and Dr. Robert Gottschalk, treasurer, re-elected.

Haralson County Hospital has been purchased by Dr. Harry H. Robinson of Charlottesville, Virginia. Dr. Robinson will have his office in the hospital building.

Dr. Raymond F. Corpe replaces Dr. Rufus Payne as director of Battey State Hospital. Dr. Payne resigned last year to accept the position of superintendent of the new state medical hospital, Augusta.

Dr. Seth J. Floyd replaces Dr. W. B. Mims, Jr., as new chief of staff at Cobb Memorial Hospital.

Dr. John T. Gentry, formerly at Communicable Disease Center, U. S. Public Health Service, Atlanta, is Alaska's first district health officer, serving as medical director of the Territory's first local health district, the new Greater Anchorage Health District.

Dr. Walter H. Thiele, formerly chief of professional services at the Veterans Administration Hospital, Fayetteville, North Carolina, succeeds Dr. Horace B. Cupp as manager of the VA hospital, Atlanta. Dr. Cupp recently was appointed manager of the new VA hospital at Durham, North Carolina.

Dr. John S. Walker is associated with Dr. Joseph H. Patterson and Dr. H. Luten Teate, Jr., Atlanta, in the practice of pediatrics.

Dr. Virgil P. Sydenstricker, Augusta, is scheduled to take part in a seminar to be conducted at the University of Edinburgh in May. He and Mrs. Sydenstricker plan to attend the coronation of Queen Elizabeth II on June 3.

Dr. D. Frank Mullins, Jr., has accepted appointment as associate professor of pathology, Medical College of Georgia, Augusta, associate pathologist to the University Hospital and also is a member of the Consultant Staff of the Veterans Hospital, Augusta.

Dr. Edward S. Marks, Marietta, has been chosen Marietta's Young Man of the Year for 1952 by the Junior Chamber of Commerce.

Dr. William Holden, Macon, left recently to visit remote sections of the Brazilian jungle in the interests of medical research.

Dr. C. A. Almand, Atlanta, has been appointed chief medical officer in the Atlanta regional office of the Veterans Administration.

Dr. Fred J. Coleman, Dublin, has been reappointed to the State Medical Board for a four-year term.

Dr. Richard B. Ewing has opened offices in the Professional Building, Macon, for the practice of dermatology.

Dr. Hiram Bush has opened offices in the DeRenne Apartments, Savannah, for the practice of allergy.

Dr. Irving L. Greenberg, Atlanta, recently received the B'nai B'rith's fifth annual "Man of the Year" award for outstanding service to his community.

KENTUCKY

Kentucky State Medical Association will hold its next annual meeting in Louisville, September 22-24.

Dr. William R. Miner, Covington, as president presided over the Southeastern Section of the American Urological Association at its seventh annual meeting held in Havana, Cuba, March 26-29. The Cuban Urological Society entertained the members of this organization with a barbecue and fiesta.

Continued on page 56

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1. McGuire et al. (1952), J. Antibiotics & Chemo., 2:281, June.
2. Heilman et al. (1952), Proc. Staff Meet. Mayo Clin., 27:385, July 16.
3. Haight and Finland (1952), New Eng. J. Med., 247:227, Aug. 14.

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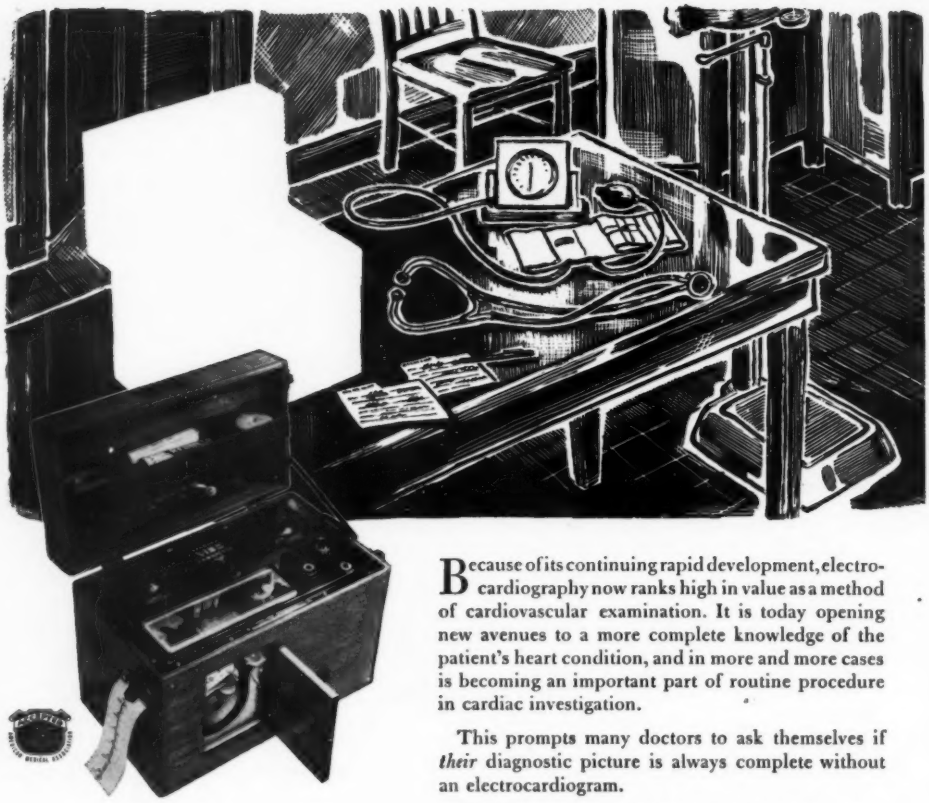

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*Crawley, G. A.: *Clinical Study of Trocinate, A New Antispasmodic Drug, M. Rec. & Ann. 43:1104, 1949.*

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Continued from page 52

Dr. John R. Prusmack, Louisville, has been appointed manager of the Veterans Administration Hospital, Palo Alta, California.

Dr. William E. Myre has returned to Paducah for the practice of medicine after completing his army service.

Dr. Charles C. Kissinger has established an office at Henderson for the practice of surgery after two years on the staff of the Veterans Hospital at Fort Wayne, Indiana.

Dr. Oris Aaron, formerly of Knoxville, Tennessee, has opened an office in West Liberty for the practice of medicine.

Dr. Charles R. Faulkner has moved from Albany to Somerset.

Dr. Charles S. Scott, Middlesboro, has gone into military service and is stationed at Fort Knox as lieutenant with the 405th Medical Group.

Dr. Harvey R. Henry, who practiced medicine in Winchester until his retirement January 1, 1945, has presented $25,000 to the Clark County Hospital's building fund. This climaxes a series of contributions to the hospital beginning in 1916 at which time he turned over to the institution all the equipment from the Henry Infirmary. Dr. Henry, a former president of Clark County Medical Society, has been a director of the hospital since its beginning forty years ago.

LOUISIANA

Louisiana State Medical Society will hold its annual meeting in New Orleans, May 7-9.

Orleans Parish Medical Society has installed Dr. Sam Hobson, president; and elected Dr. J. O. Weilbaecher, president-elect; Dr. Howard Mahorner, first vice-president; Dr. John J. Irwin, second vice-president; Dr. A. N. Houston, third vice-president; Dr. Charles B. Odom, secretary; and Dr. Felix Planche, treasurer.

Eye, Ear, Nose and Throat Hospital, New Orleans, has elected to its staff Dr. William M. Boles, president; Dr. Carl Granberry, first vice-president; Dr. Harold Tabb, second vice-president; and Dr. Samuel Zurik, secretary-treasurer.

Hotel Dieu, New Orleans, has elected to its medical staff Dr. Hugh T. Beacham, president; Dr. L. Sidney Charbonnet, Jr., vice-president; and Dr. Nick Accardo, secretary-treasurer.

Mercy Hospital, New Orleans, has elected to its staff Dr. Edwin L. Zander, president; Dr. Joseph A. Vella, vice-president; Dr. Esmond A. Fatter, secretary; and Dr. O'Neil L. Pollingue, treasurer.

Dr. Louis Ochs, Jr., clinical assistant professor of medicine, Louisiana State University School of Medicine, New Orleans, has been named chief of gastroenterology at Touro Infirmary.

Dr. George E. Burch, professor and chairman of the department of medicine, Tulane University School of Medicine, New Orleans, has been chosen by *Modern Medicine* magazine as one of ten outstanding recent contributors to medical knowledge.

Tulane University School of Medicine, New Orleans, will make two important studies in polio with the two March of Dimes grants totaling $87,978: one grant of $75,765 for a five-year period with $37,865 allocated for the first year will be used for a comprehensive study of the circumstances under which individuals become infected with polio virus; and the second grant of $12,213 for continuation of investigations of the relations of Coxsackie virus infection to human polio and other virus diseases.

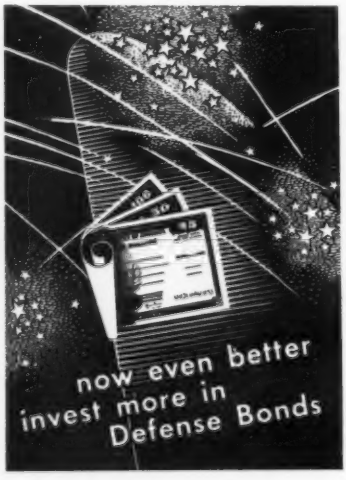
Dr. Edward L. King, professor emeritus of obstetrics, Dr. Conrad G. Collins, professor of obstetrics and gynecology, and Dr. B. Bernard Weinstein, associate professor of clinical gynecology, Tulane University School of Medicine, New Orleans, have been awarded special diplomas by the consul-general of Venezuela at New Orleans, for contributions in internal medicine.

MARYLAND

Dr. Edwards A. Park, emeritus professor of pediatrics, Johns Hopkins University School of Medicine, Baltimore, has served for years on the editorial board of the *Journal of Pediatrics*. The December 1952 issue of this journal was a *Festschrift* in honor of Dr. Park on his seventy-fifth birthday.

Dr. Arthur Kornberg, chief of the enzyme and metabolism section of the National Institutes of Health, Bethesda, since 1948, has been appointed professor of microbiology at Washington University School of Medicine, St. Louis, Missouri, where he previously served as research assistant in biological chemistry.

Continued on page 58



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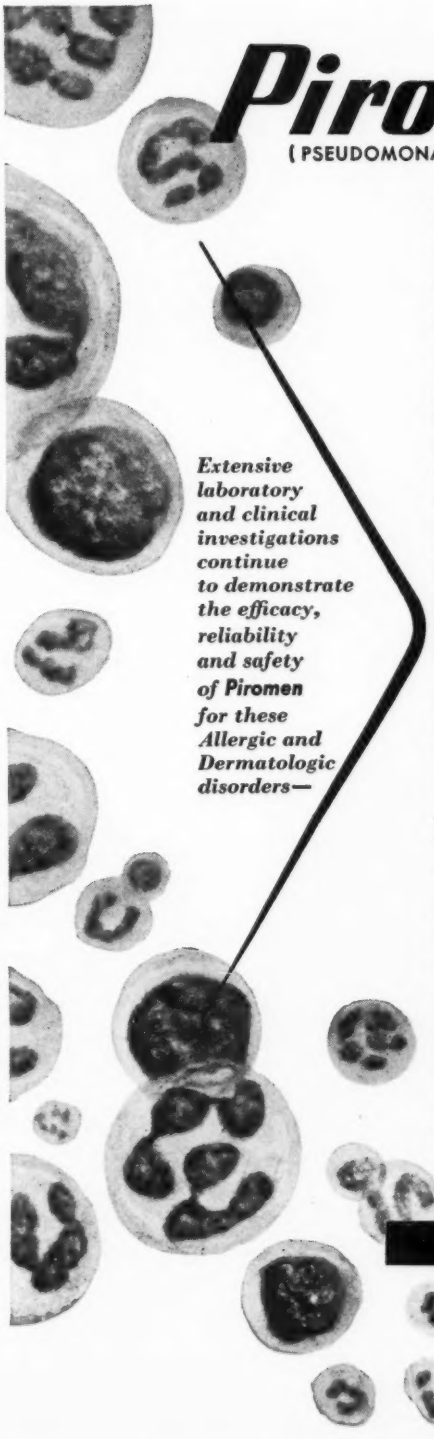

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Continued from page 56

MISSISSIPPI

Mississippi State Medical Association will hold its 85th annual meeting in Biloxi, May 12-14. House of Delegates will meet May 11.

The new $8,500,000 University Medical School and Teaching Hospital is under construction in Jackson and completion of the project is scheduled for 1954. This will give the state its first four-year medical course under the supervision of the University of Mississippi School of Medicine. The teaching hospital will be Mississippi's largest, with 350 beds and facilities for the care of 50,000 outpatients annually, and eventually expansion to 525 beds is planned. The school will care for 320 students, and possibly an eventual enrollment of 400.

Dr. Hugh Lawson McKinnon, Hattiesburg, will be Mississippi's guest of honor at the First Western Hemisphere Conference of the World Medical Association to be held in Richmond, April 23-25, in observance of the lengthening of life and the constant improvement of human health.

Dr. Charles M. Murry, Oxford, has been named the "Man of Oxford for 1952."

Dr. David B. Wilson, a native of Yazoo City and who has been with the Mississippi Commission on Hospital Care since 1951, has been made director of the 350-bed University teaching hospital under construction in Jackson. He will help in supervising the construction, the purchase and installation of hospital equipment for the new hospital.

MISSOURI

The Southwest Allergy Forum will meet in Kansas City, Hotel Muehlebach, Kansas City, June 14-16, under the presidency of Dr. Orval Withers.

Midwest Hospital Association will hold its meeting in Kansas City, Municipal Auditorium, April 15-17.

American Radium Society will meet in St. Louis, Chase Hotel, April 19-22.

Missouri Tuberculosis Association will meet in Kansas City, Hotel President, April 24-25.

Missouri Public Health Association will meet in Jefferson City, Hotel Governor, May 4-6.

Catholic Hospital Association will meet in Kansas City, Municipal Auditorium, May 25-28.

Perry County Memorial Hospital has elected to its medical staff Dr. William Utterman, president; Dr. L. W. Feltz, vice-president; and Dr. J. J. Bredall, secretary.

Dr. August A. Werner, St. Louis, served as an officer in the section on endocrinology at the International Cruise-Congress to South America and the West Indies, leaving New York in January. He spoke before the Congress on "Climacteric in Women and Men."

Continued on page 60

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WANTED—For October 1, Physician for Alabama State Mental Institution. Salary $6,000.00 to $7,800.00. Must be Licensed. Write Dr. J. S. Tarwater, Superintendent The Bryce Hospital, Tuscaloosa, Alabama.

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CREAM OR OINTMENT

Continued from page 58

Dr. J. M. Perry, Princeton, was honored recently for his fifty years of practice at a dinner given by the Princeton Chamber of Commerce.

St. Louis University School of Medicine will receive $101,380 under the will of Dr. William Washington Graves, director of the department of neuropsychiatry for 23 years, who died in April 1949.

Dr. Ira H. Lockwood, Kansas City, was installed president of the Radiological Society of North America at the 38th annual meeting held in Cincinnati, Ohio, recently; and Dr. C. Edgar Virden, Kansas City, was elected a member of the board of directors.

NORTH CAROLINA

Medical Society of the State of North Carolina will hold its next annual meeting in Pinehurst, May 10-13.

The Association of Southeastern Biologists will hold its fourteenth annual meeting at the University of North Carolina, Chapel Hill, April 16-18, under the presidency of Dr. Margaret Hess, Rock Hill, South Carolina. Dr. C. S. Shoup, Oak Ridge, Tennessee, is vice-president; Dr. Bruce D. Reynolds, Charlottesville, Virginia, president-elect; and Dr. Mary Esther Gaulden, Oak Ridge, Tennessee, secretary-treasurer.

Charlotte Ophthalmological Society was formed recently and the following officers elected: Dr. Herbert C. Neblett, president; Dr. James D. Stratton, vice-president; and Dr. Thomas D. Ghent, secretary and treasurer.

OKLAHOMA

The Tulsa Child Guidance Clinic was moved to the Junior League Children's Convalescent Hospital on March 1 and is housed in the new wing of the hospital. Dr. Paul C. Benton, director of the Child Guidance Clinic, has also been named medical director of the Junior League Children's Hospital.

Dr. R. M. Howard, Oklahoma City, has been selected Oklahoma's guest of honor at the First Western Hemisphere Con-

Continued on page 62

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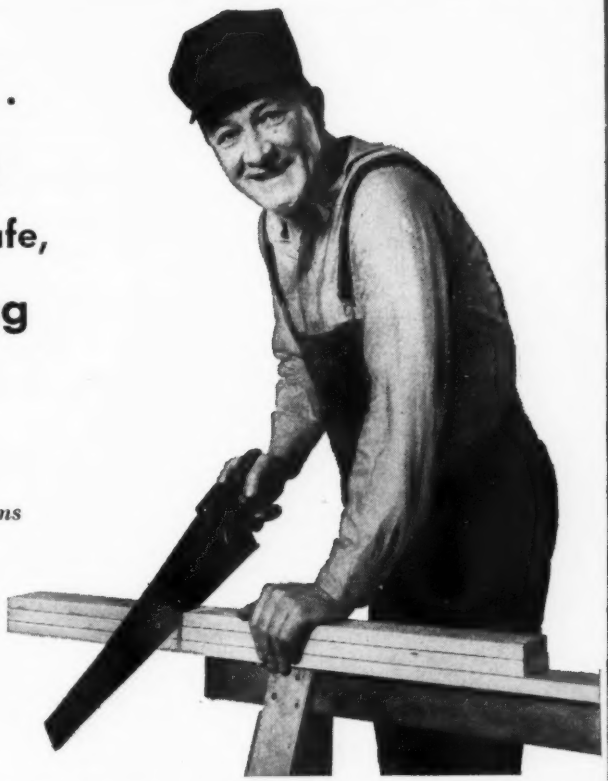

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RAHWAY, NEW JERSEY

Continued from page 60

ference of the World Medical Association in Richmond, Virginia, April 23-25, with all expenses paid by the A. H. Robins Company, a pharmaceutical firm founded in Richmond seventy-five years ago.

Dr. M. H. Newman, Shattuck, has been appointed to the Board of Medical Examiners, succeeding his father, Dr. O. C. Newman, who resigned for the unexpired term.

Dr. John B. Jacob, Waurika, has been recalled to active duty with the navy and has reported to Memphis, Tennessee.

Dr. J. M. Newton, formerly of Massachusetts, has been named medical director for the Oklahoma State Hospital, Clinton.

Dr. D. D. Cramblett has moved from Stigler to Holdenville.

Dr. Ray J. Camp, Woodward, is taking a three-year surgical residency in Minneapolis, Minnesota.

Dr. Roger Reid, Ardmore, has been elected president of the staff of Hardy Sanitarium, Ardmore.

Dr. M. Wilson Mahone, Hobart, has been named part-time medical consultant for the Kiowa County Health Department.

SOUTH CAROLINA

South Carolina Medical Association will hold its next annual meeting in Columbia, May 12-14.

Dr. Kenneth M. Lynch, Charleston, was honored at the recent Founder's Day Banquet held in Charleston when his portrait was presented to the Medical College of South Carolina by his associates and former students. The portrait will be hung in the foyer of the new teaching school. Dr. Lynch served as professor of pathology and was also dean of the school from 1943 to 1948 and is now president of the institution.

Dr. Robert A. Brown, recent consultant to the United Air Forces in Europe and a diplomate, American Board of Ophthalmology, became associated on February 1 with Dr. J. W. Jervey and Dr. A. J. Goforth, Jr., Greenville.

A portrait of the late Dr. N. B. Heyward was unveiled recently in the Lily Hardin Nurses Home of the Baptist Hospital, Columbia. Dr. Heyward served many years as secretary of the South Carolina State Board of Medical Examiners and also for two years as secretary of the South Carolina Medical Association.

Dr. W. H. Muller, a native of Dillon and now associate professor of medicine, University of California of Los Angeles School of Medicine, was selected as one of the ten outstanding men of 1952 by the U. S. Junior Chamber of Commerce.

Christie Pediatric Group has been organized in Greenville by Drs. R. C. Brownlee, W. R. DeLoache, H. P. Jackson and W. B. Mills.

Dr. H. Neils Wessel has opened offices in Greenwood for the practice of urology.

Dr. Clyde Higgins, Jr., has resumed practice in Easley after receiving his discharge from the U. S. Navy.

Dr. A. C. Magee, Cheraw, has been recalled to active duty with the United States Air Force.

Dr. W. C. Thompson, after serving forty years as a general practitioner in Dublin, Georgia, and several years of retirement, has resumed his practice in the Flat Rock community of Anderson County.

Dr. Frank Kitchen, a retired Greenville city health officer, as a token of appreciation for his long and efficient service in Greenville, was recently presented with a deep freeze unit at a banquet tendered by dairymen in that part of the state.

Dr. J. E. McLaughlin has opened offices in Ehrhardt for the practice of medicine.

Dr. Robert W. Houseal has been named Chief of Professional Services at the Veterans' Hospital, Columbia. Dr. Houseal is a native of Newberry and practiced medicine there from 1924 to 1942.

Dr. Roy P. Finney, Jr., son of Dr. Roy Finney of Spartanburg, has been granted a $3,600 fellowship by the National Cancer Institute and is now serving his internship at the Brady Institute, Johns Hopkins Hospital, Baltimore, Maryland, and will carry on his research there.

Dr. Horace Whitworth, Greenville, has been named president of the staff of the St. Francis Hospital for the year 1953. Elected to the newly organized Associate Medical Staff: Dr. Jack Kane, chairman; Dr. Peter Gazes, vice-chairman; and Dr. H. H. Addlestone, secretary.

The Medico-Chirurgical Club, Charleston, has elected Dr. Leon Banov, Jr., president; Dr. H. H. Addlestone, vice-

Continued on page 64

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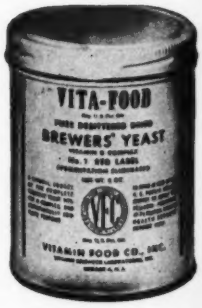

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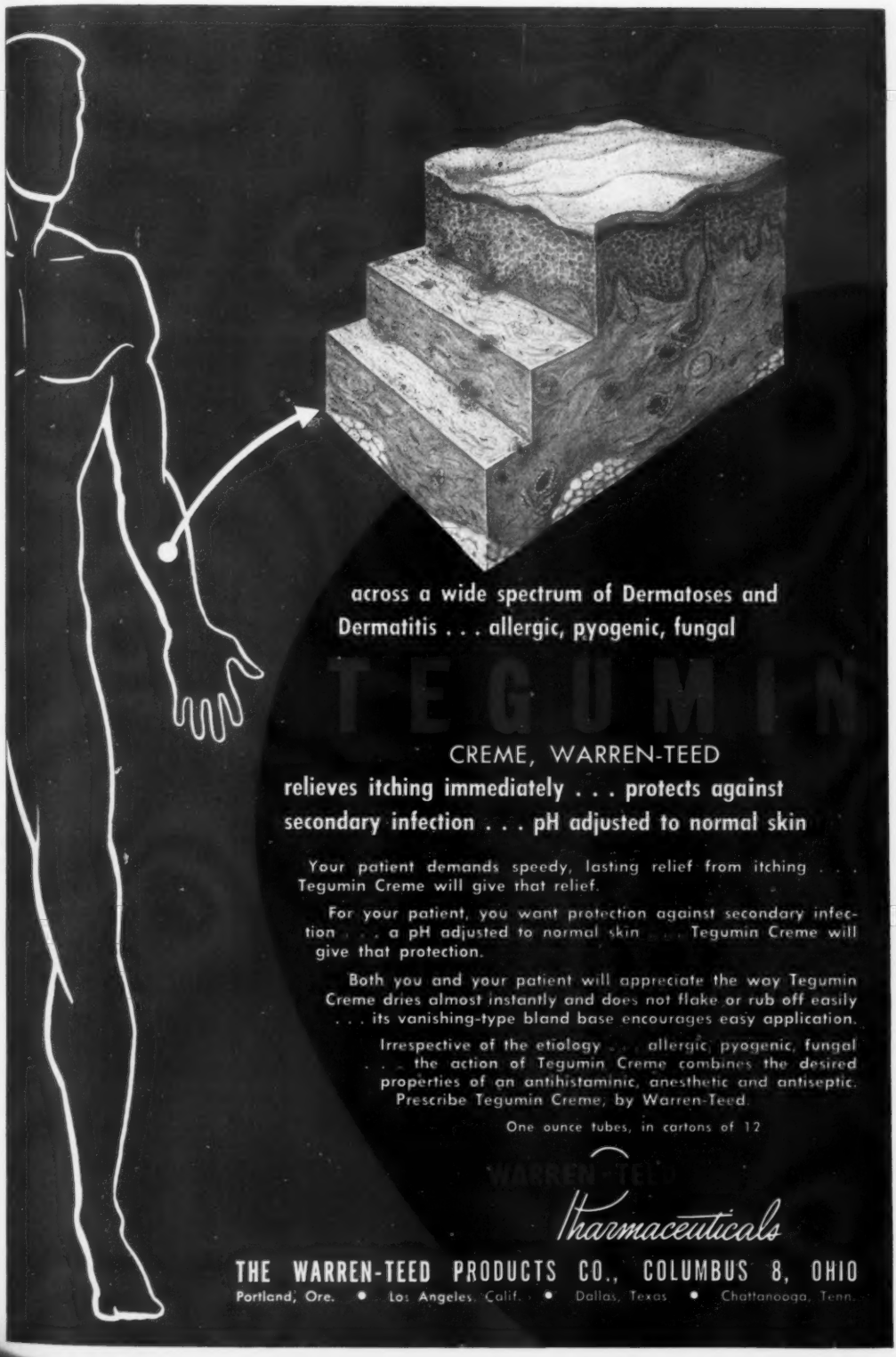
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Continued from page 62

president; Dr. Kenneth Lynch, Jr., secretary-treasurer; and on the Board of Censors, Dr. C. Capers Smith, Dr. D. L. Maguire, Jr., and Dr. I. Grier Linton.

TENNESSEE

The Southeastern Allergy Association will hold its annual meeting in Nashville, Andrew Jackson Hotel, May 15-16.

An annual lectureship has been established at Vanderbilt University School of Medicine, Nashville, through the generosity of an alumnus, in memory of Dr. Barney Brooks, the late professor of surgery. The first lecture was given in January by Dr. Evarts A. Graham, St. Louis, Missouri.

Dr. Charles H. Heacock, Memphis, was elected first vice-president of the Radiological Society of North America at its 38th annual meeting held in Cincinnati, Ohio, recently.

Shelby County Medical Society has installed Dr. E. G. Campbell, president; and elected Dr. S. F. Strain, president-elect; Dr. J. R. Reinberger, vice-president; Dr. Jean M. Hawkes, secretary; Dr. Battle Malone, II, treasurer; and Mr. Robert C. Bird, executive secretary, all of Memphis.

Dr. R. R. Overman, Department of Physiology, University of Tennessee College of Medicine, Memphis, will attend a meeting in Istanbul, Turkey, August 28-September 4 and speak at the fifth International Congresses on Tropical Disease and Malaria on the relation of the function of the adrenal gland to malaria and to post-malaria debility, the report being based on research conducted at the University from 1945 to 1950. This trip will be made possible by a grant of $1,275 awarded to Dr. Overman by the National Science Foundation.

The University of Tennessee Divisions of Medicine and Pathology and Bacteriology, in cooperation with the John Gaston Hospital, Memphis, are conducting a study of effects of small doses of aureomycin on persons more than fifty years old. The study is financed by a research grant of $24,000 awarded by the American Cyanamid Company, Lederle Laboratories Division, Pearl River, New York.

Dr. N. B. Williams, a native of Andersonville, has opened offices for the practice of medicine in Clinton, following two years of military service.

Dr. M. J. Adams, Kingsport, has been named president of the Holston Valley Community Hospital.

Dr. S. J. Sullivan, Cleveland, is chief of staff of the new Bradley County Memorial Hospital.

Dr. William I. Proffitt, formerly of Cookeville and former resident in surgery at Erlanger Hospital, is associated with Dr. Madison S. Trewhitt, Cleveland.

Dr. L. D. Courtnor, after two years in the armed services, has returned for the practice of medicine in Athens.

Dr. W. J. Fedack, Copperhill, is associated with the Martin-Riser Clinic, Pell City, Alabama.

Dr. Leon Goforth is associated with Dr. C. C. Johnson, Rogersville, in the practice of medicine.

Dr. A. W. Diddle is the new chief of staff at Knoxville Baptist Hospital. Dr. John Range is vice-chief, and Dr. Robert Newman, secretary.

Dr. E. Bearden Cunningham has opened an office in Blountville for three days weekly but will maintain his offices in Bristol where he has practiced since 1951.

Dr. Reece Bennett DeBerry is associated with Dr. J. W. Carroll, Savannah.

Dr. Bruce Galbraith and Dr. Claude C. Snoody have opened offices together at Tullahoma.

Dr. J. H. Gammon, Knoxville, will be Tennessee's guest of honor at the First Western Hemisphere Conference of the World Medical Association in Richmond, Virginia, April 23-25.

Mid-South Postgraduate Medical Assembly (Tennessee, Arkansas and Mississippi) at its annual meeting held in Memphis in February installed Dr. L. S. Nease, Newport, president; and elected Dr. R. B. Caldwell, Baldwyn, Mississippi, president-elect; Dr. N. L. Hyatt, Covington, vice-president for Tennessee; Dr. George A. Brown, Water Valley, vice-president for Mississippi; and Dr. F. S. Dozier, Marianna, vice-president for Arkansas. The new president-elect is selected from a different state each year.

Dr. Joseph B. Parker, Jr., associate professor of psychiatry, University of Tennessee College of Medicine, Memphis, resigned March 31 to join the staff of Duke University School of Medicine, Durham, North Carolina.

Dr. James A. Taylor, senior psychiatrist at Kennedy Veterans' Hospital, Memphis, joined the staff of the University of Tennessee College of Medicine on March 1. Dr. Taylor will be

Continued on page 66

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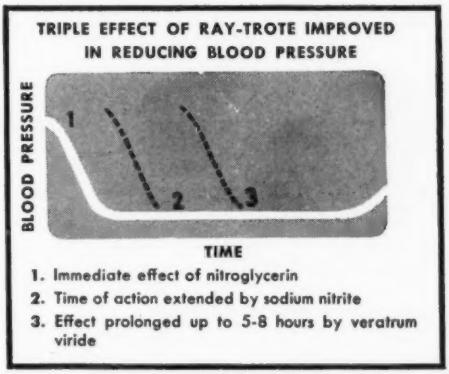
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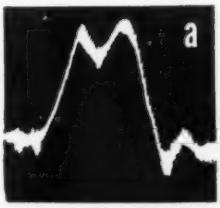

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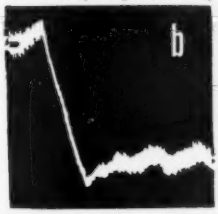

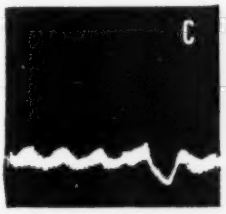

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Continued from page 64

assistant professor of psychiatry and neurology and clinical director at the Gailor Psychiatric Hospital.

Dr. A. Lynn Herring and Dr. Howell D. Woodson have announced their association for the practice of urology and urological surgery with offices in the Baptist Hospital Building, Memphis.

TEXAS

Postgraduate Medical Assembly of South Texas will meet in Houston, July 20-22.

Texas Division, American Cancer Society, held its seventh annual meeting in Austin in January and elected Mr. Travis Wallace, Dallas, president; Dr. G. V. Brindley, Temple, vice-president for medical activities; Mrs. Joseph M. Perkins, Temple, vice-president for medical activities; Mrs. Joseph M. Perkins, Eastland, vice-president for lay activities; and Mr. Frank C. Smith, Houston, chairman of the board of directors.

Dr. D. D. Duncan, professor of anatomy, University of Texas Medical Branch, Galveston, has been named associate dean in charge of the graduate school of the Medical Branch.

Dr. G. R. Herrman, Galveston, was awarded an honorary professorship at Santiago, Chile, and membership in the Perma Cardiological Society, Lima, Peru, during a recent lecture tour to South America.

Dr. J. J. Shiller, Rowena, was honored by his community for forty years of service, receiving a plaque and certificate of merit from the Woodmen of the World.

The Victor Hexter Memorial Lectureship Fund in psychiatry has been established at Southwestern Medical School of the University of Texas, Dallas, by a gift of $1,000 from Louis J. Hexter in memory of his father, Victor Hexter. The fund will be used to bring outstanding teachers of psychiatry to the school.

Dr. A. W. B. Cunningham, formerly of the University of Edinburgh, has been appointed professor of pathology, University of Texas Medical Branch, Galveston, and will be responsible for instruction and research on arthritis and diseases of the joints.

VIRGINIA

American Association of Genito-Urinary Surgeons will meet in Hot Springs, May 6-8, under the presidency of Dr. A. J. Scholl, Los Angeles, California.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons will meet in Hot Springs, September 10-12, under the presidency of Dr. Nicholson J. Eastman, Baltimore, Maryland.

American Ophthalmological Society will meet at Hot Springs, May 28-30, under the presidency of Dr. Conrad Berens, New York City.

Richmond Academy of Medicine has installed Dr. R. G. McAllister, president; and elected Dr. W. Linwood Ball, president-elect; Dr. Theodore Kohn, vice-president; Dr. F. E. Oglesby, secretary; and Dr. Harold I. Nemuth, treasurer. Dr. Richard Crossen and Dr. H. Mason Wingfield are the two new directors.

Dr. Richard J. Ackart, director of the University of Virginia Hospital, became executive director of the Virginia Hospital Service Association (Blue Cross) on February 1.

Richmond Eye, Ear, Nose and Throat Society has elected Dr. J. Warren Montague, president, and re-elected Dr. Charles N. Romaine, secretary-treasurer.

WEST VIRGINIA

West Virginia State Medical Association will hold its eighty-sixth annual meeting in White Sulphur Springs, The Greenbrier, July 23-25.

Dr. Emmet D. Moyers has moved from Norton to Widen where he will continue in industrial practice for the Elk River Coal and Lumber Company.

Dr. S. W. Jabaut, Shinnston, has retired from active practice. He has accepted appointment as health officer for the district comprised of Greenbrier, Pocahontas and Monroe counties and will assume his new duties with headquarters at Lewisburg after he completes his work for Master's Degree in Public Health at the School of Public Health, Chapel Hill, North Carolina.

Dr. R. W. Brown, Spencer, has been called to active duty in the Medical Corps of the Army and is stationed at Fort Sam Houston, Texas.

Dr. James S. Kessell has resumed practice at Ripley after being released from military service.

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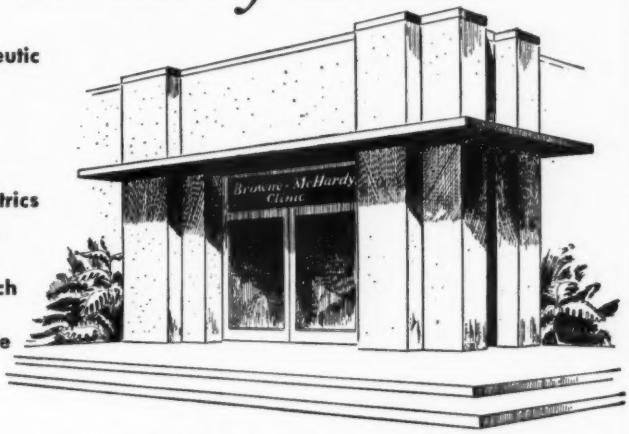

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1. Heimark, J.J. and Parsons, R.L.: Minnesota Med. 33:1102, Nov. 1950

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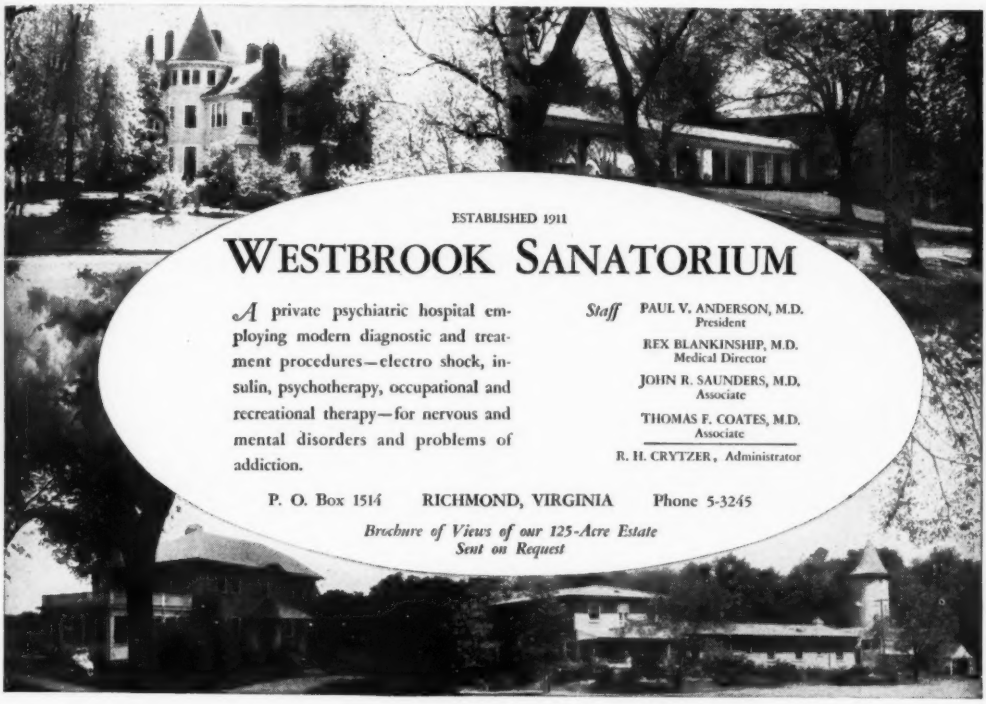
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1. Frost, L. H., and Jackson, R. L.: J. Pediat. 39: 585-592, 1951.

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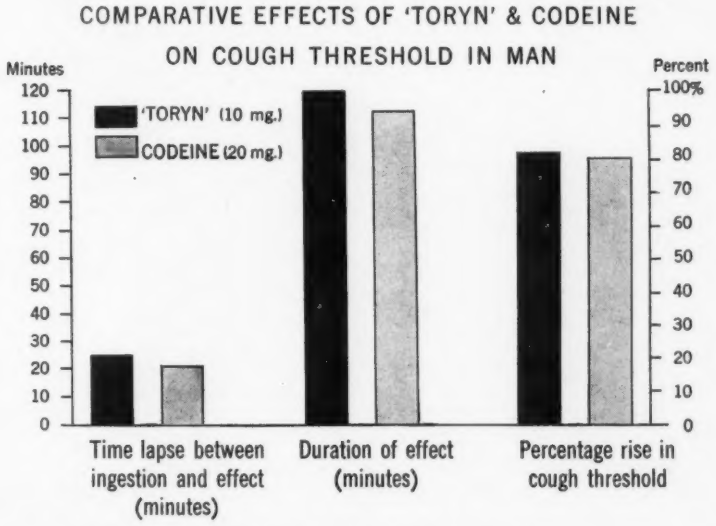

1. Blix, M.: On the Antitussive Effect of (β-diethylaminoethyl 1-phenyl-cyclopentane-1-carboxylate) ethanedisulfonate, S.K.F. ("Toryn"). *In manuscript.*

†(Average percentage rise in cough threshold) × (duration of activity)

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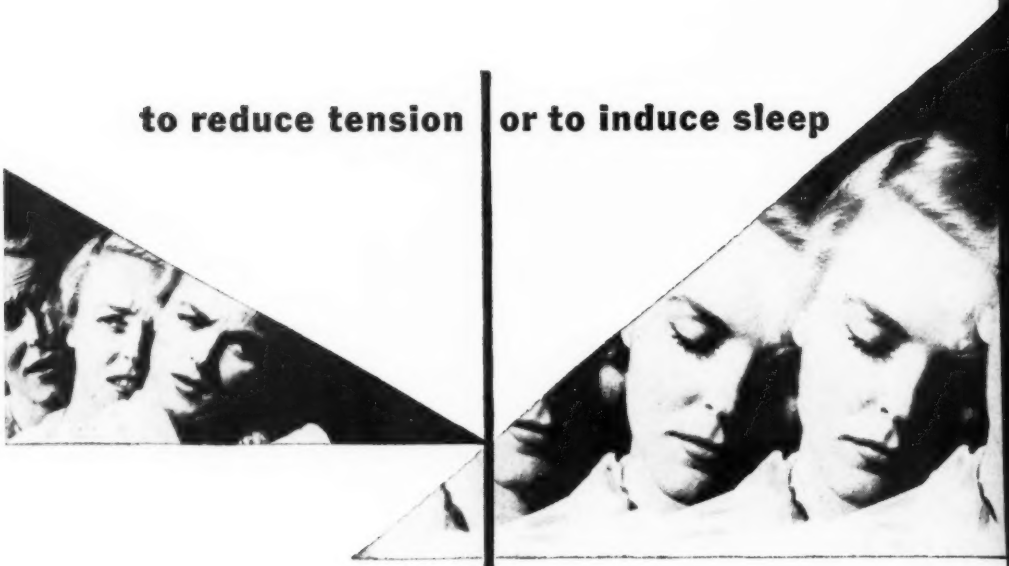

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